(FILE 'HOME' ENTERED AT 17:38:53 ON 04 MAR 2002)

FILE 'BIOSIS, CABA, CAPLUS, EMBASE, LIFESCI, MEDLINE, SCISEARCH, USPATFULL, JAPIO' ENTERED AT 17:39:03 ON 04 MAR 2002

L1 3 S DETECTING COLITIS
L2 8 S DIAGNOSING COLITIS

=>

L8 ANSWER 1 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:316901 BIOSIS
DN PREV200100316901
TI Obliterative arteritis with nitric oxide synthase and HLA-DR expression in Crohn's **colitis**.
AU Yokoyama, Kaoru; Mitomi, Hiroyuki (1); Kobayashi, Kiyonori; Katsumata, Tomoe; Saigenji, Katsunori; **Okayasu, Isao**
CS (1) Department of Pathology, School of Medicine, Kitasato University East Hospital, 2-1-1 Asamizo-dai, Sagamihara, Kanagawa, 228-8520 Japan
SO Hepato-Gastroenterology, (March April, 2001) Vol. 48, No. 38, pp. 401-407. print.
ISSN: 0172-6390.
DT Article
LA English
SL English
AB Background/Aims: To cast light on whether inflammatory vascular injury is a possible pathogenic mechanism in Crohn's disease, the histological characteristics of vascular lesions were investigated. Methodology: Affected vessels in surgically resected colons from 23 patients with Crohn's disease, 20 with ulcerative **colitis**, 7 with ischemic **colitis**, and 9 normal controls were analyzed by Victoria blue and hematoxylin and eosin staining as well as immunohistochemistry for HLA-DR, nitric oxide synthase, vascular endothelial growth factor and E-cadherin. Results: Inflammatory-cell infiltrates affecting arteries, accompanied by obliterative intimal thickening, were more frequent in Crohn's disease cases than in the other groups ($P<0.05$-0.0001). Crohn's disease activity was positively correlated with the degree of obliterative arteritis. Granulomatous vasculitis was found exclusively in Crohn's disease (10 cases; 43.5%). In addition, focally enhanced endothelial staining of HLA-DR, with expression in granulomas adjacent to vessels was occasionally observed. In the endothelium of affected vessels, strong expression of HLA-DR was more prevalent in Crohn's disease and/or ulcerative **colitis** as compared with the ischemic **colitis** and controls ($P<0.05$-0.01). In the involved arteries, enhanced endothelial nitric oxide synthase expression was most common in Crohn's disease among the groups ($P<0.05$). A few cases of Crohn's disease, ulcerative **colitis** and ischemic **colitis** were positive for inducible nitric oxide synthase, vascular endothelial growth factor or E-cadherin in the vessel walls. Conclusions: The presence of characteristics obliterative arteritis and granulomatous vasculitis, a possible cause of ischemic injury, supports, in part, a vascular hypothesis for the pathogenesis of Crohn's disease. Enhanced expression of endothelial nitric oxide synthase and HLA-DR possibly reflects compensatory endothelium-mediated vasolidation and amplification of the immune response, respectively.

L8 ANSWER 2 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2001:124391 BIOSIS
DN PREV200100124391
TI Polysaccharides extracted from human tubercle bacilli (specific substance of Maruyama) reduces carcinogenesis in murine ulcerative **colitis**.
AU Sakamoto, Shinobu (1); **Okayasu, Isao**; Iida, Kazumi; Fujita, Keishirou; Yarimizu, Takashi; Nagasawa, Hiroshi
CS (1) Medical Research Institute, Tokyo Medical and Dental University, 1-5-45, Yushima, Bunkyo-ku, Tokyo, 113-8510: motoend@mri.tmd.ac.jp Japan
SO Anticancer Research, (November December, 2000) Vol. 20, No. 6B, pp. 4295-4300. print.
ISSN: 0250-7005.
DT Article
LA English
SL English
AB Polysaccharides extracted from human tubercle bacilli (specific substance of Maruyama) have been clinically applied in patients with malignant

diseases in Japan and other countries. It is known that increased colorectal carcinogenesis occurs in patients with ulcerative **colitis**. The repeated mucosal necrosis-regeneration sequence in chronic ulcerative **colitis** induced with 3 % dextran sulfate sodium led to colorectal carcinogenesis in azoxymethane-pretreated mice. Simultaneously multiple injections with the polysaccharides reduced the increases in thymidylate synthase and thymidine kinase activities and a number of bromodeoxyuridine-incorporated S-phase cells in colorectal tissues resulted in the reduction of tumorous regions with high-grade dysplasia.

L8 ANSWER 3 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:458194 BIOSIS
DN PREV200000458194
TI Decreased expression of CD44, alpha-catenin, and deleted colon carcinoma and altered expression of beta-catenin in ulcerative **colitis** -associated dysplasia and carcinoma, as compared with sporadic colon neoplasms.
AU Mikami, Tetuo (1); Mitomi, Hiroyuki; Hara, Atsuko; Yanagisawa, Nobuyuki; Yoshida, Tsutomu; Tsuruta, Osamu; **Okayasu, Isao**
CS (1) Department of Pathology, School of Medicine, Kitasato University, 1-15-1 Kitasato, Sagamihara-si, Kanagawa, 228-8555 Japan
SO Cancer, (August 15, 2000) Vol. 89, No. 4, pp. 733-740. print. ISSN: 0008-543X.
DT Article
LA English
SL English
AB BACKGROUND: To clarify the cell adhesion status in ulcerative **colitis** (UC)-associated colon neoplasm, expression of cell adhesion molecules were investigated and compared with that of sporadic colon neoplasm. METHODS: A total of 14 low grade dysplasias, 16 high grade dysplasias, and 8 adenocarcinomas associated with UC and 17 sporadic adenomas with mild to moderate dysplasia, 22 adenomas with severe dysplasia, and 15 invasive adenocarcinomas were immunohistochemically examined using monoclonal antibodies against CD44, E-cadherin, alpha- and beta-catenin, and deleted colon carcinoma (DCC). RESULTS: CD44, especially its standard form, and DCC expression was stronger in the sporadic colon neoplasms than in the UC-associated lesions. Although E-cadherin did not show significant differences between the two cases, alpha-catenin was more expressed in sporadic colon adenomas with severe dysplasia and carcinomas than in their UC-associated counterparts. Membranous beta-catenin staining was stronger in UC-associated neoplasms, whereas sporadic lesions had greater cytoplasmic and nuclear expression. CONCLUSIONS: The differences in cell adhesion molecule expression suggests that UC-associated and sporadic colon neoplasms arise from different pathways of tumorigenesis.

L8 ANSWER 4 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 2000:257842 BIOSIS
DN PREV200000257842
TI Significant increase in prostaglandin E-main urinary metabolite by laxative administration: Comparison with ulcerative **colitis**.
AU Fujiwara, Mutsunori (1); **Okayasu, Isao**; Oritsu, Masae; Komatsu, Junko; Yoshitsugu, Michiyasu; Katoh, Yoshihisa; Bandoh, Takafumi; Toyoshima, Hiroshi; Kase, Yoshio; Sugihara, Kunio; Kanno, Jun; Hayashi, Yuzo
CS (1) Department of Clinical Pathology, Japanese Red Cross Medical Center, Hiroo 4-1-22, Shibuya-ku, Tokyo, 150-0012 Japan
SO Digestion, (July 26, 2000) Vol. 61, No. 3, pp. 201-206. print.. ISSN: 0012-2823.
DT Article
LA English
SL English
AB Objective: To assess the production of prostaglandin E2, an important chemical mediator in diarrhea induced by laxative administration, a

prostaglandin E-main urinary metabolite (7alpha-hydroxy-5,11-diketotetranor-prosta-1,16-dioic acid, PGE-MUM) was measured in healthy volunteers and compared with the values of patients with ulcerative **colitis**. Methods: PGE-MUM was determined by a simplified immunoassay of bicyclic PGE-MUM and analyzed for the influence of laxative administration and active/remission phases of ulcerative **colitis**. Results: Administration of laxatives induced a significant increase in PGE-MUM in healthy volunteers. A significant elevation was also found in the active as compared with the remission phase of ulcerative **colitis**. The PGE-MUM levels were significantly correlated with our modified Talstad scores, clinical disease activity indices in ulcerative **colitis**. It was confirmed by time course studies of individual patients that changes in PGE-MUM correlated well with **colitis** activity. Conclusion: Laxative administration induces production of prostaglandin E2 as one of the chemical mediators, although its production grade is relatively low as compared with ulcerative **colitis** in the active phase.

L8 ANSWER 5 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:332009 BIOSIS
DN PREV199900332009
TI Enhanced epithelial cell turnover associated with p53 accumulation and high p21WAF1/CIP1 expression in ulcerative **colitis**.
AU Arai, Nobuyasu; Mitomi, Hiroyuki (1); Ohtani, Yoshimasa; Igarashi, Masahiro; Kakita, Akira; **Okayasu, Isao**
CS (1) Department of Pathology, School of Medicine, Kitasato University East Hospital, 2-1-1 Asamizodai, Sagamihara, Kanagawa, 228-8520 Japan
SO Modern Pathology, (June, 1999) Vol. 12, No. 6, pp. 604-611.
ISSN: 0893-3952.
DT Article
LA English
SL English
AB To cast light on accelerated epithelial cell turnover as an important risk factor of dysplasia and carcinoma development in patients with long-standing ulcerative **colitis** (UC), we examined cell proliferation and cell death, as well as expression of apoptosis-related markers, including p53 and p21WAF1/CIP1, in a series of cases. Biopsy specimens (n = 176; 84, active phase; 92, remission) were endoscopically obtained from 25 Japanese patients with UC. As controls, 68 biopsy specimens of normal mucosa were also examined from 27 Japanese patients with colon polyps. We counted the numbers of mitoses, apoptotic bodies, Ki-67-immunoreactive cells, and p21WAF1/CIP1-immunoreactive cells per 1000 crypt cells and the numbers of p53-positive cells per crypt. All of the indices in active UC were significantly higher than in either remitting UC cases or normal cases (mean mitotic index = 0.52, 0.28, and 0.15%, respectively; apoptotic index = 5.40, 2.91, and 1.30%, respectively; Ki-67 labeling index = 39.5, 28.3, and 26.8%, respectively; p21WAF1/CIP1 labeling index = 33.6, 20.0, and 19.0%, respectively; p53 labeling index = 0.66, 0.13, 0.13 per crypt, respectively). In addition, the mitotic, apoptotic, and Ki-67 labeling indices were increased in remitting UC of more than 10 years' duration, in comparison with those of less than 10 years' duration or the normal group. Immunostaining of serial sections revealed a small number of crypt cells co-expressing p53 and p21WAF1/CIP1. Increases in both epithelial cell proliferation and cell death, partially associated with p53 accumulation and high p21WAF1/CIP1 expression, are thus characteristic of active phase UC, as well as in remission of longstanding UC. Accelerated epithelial cell turnover caused by chronic inflammation and epithelial damage might predispose the mucosa to DNA damage, resulting in an elevated risk of mutation in line with dysplasia and carcinoma development in patients withUC.

L8 ANSWER 6 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:258517 BIOSIS
DN PREV199900258517

TI Effect of Icatibant, a bradykinin B2 receptor antagonist, on the development of experimental ulcerative **colitis** in mice.
AU Arai, Yoshinori (1); Takanashi, Hitoshi; Kitagawa, Hiroshi; Wirth, Klaus J.; **Okayasu, Isao**
CS (1) Laboratory for Pharmacology, Lead Optimization, Drug Innovation and Approval, Hoechst Marion Roussel Ltd., 1-3-2 Minamidai, Kawagoe, Saitama, 350-1165 Japan
SO Digestive Diseases and Sciences, (April, 1999) Vol. 44, No. 4, pp. 845-851.
ISSN: 0163-2116.
DT Article
LA English
SL English
AB Dextran sulfate sodium-induced **colitis** in mice has been recognized as a model for human ulcerative **colitis**. Using this model, we carried out a study on the preventive effect of Icatibant, a bradykinin B2 receptor antagonist previously called HOE 140, on the development of **colitis**. Subcutaneous administration of Icatibant (0.3 or 1.5 mg/kg) significantly suppressed shortening of the large intestine and worsening of the general health. Oral administration of Icatibant (50 mg/kg) significantly suppressed shortening of the large intestine, the onset of diarrhea, and worsening of the general health. In addition, the oral treatment significantly inhibited the development of **colitis** that was observed histopathologically. These results indicate a role of BK in the development of dextran sulfate sodium-induced **colitis** in mice, and suggest that BK could be important in human ulcerative **colitis**.

L8 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:147366 BIOSIS
DN PREV199900147366
TI **Colitis** in chronic granulomatous disease resembling Crohn's disease: Comparative analysis of CD68-positive cells between two disease entitites.
AU Mitomi, Hiroyuki (1); Mikami, Tetsuo; Takahashi, Hiroyuki; Igarashi, Masahiro; Katsumata, Tomoe; Ihara, Atsushi; Ohtani, Yoshimasa; Ohta, Takeo; **Okayasu, Isao**
CS (1) Dep. Pathol., Sch. Med., Kitasato Univ. E. Hospital, 2-1-1 Asamizo-dai, Sagamihara, Kanagawa 228 Japan
SO Digestive Diseases and Sciences, (Feb., 1999) Vol. 44, No. 2, pp. 452-456.
ISSN: 0163-2116.
DT Article
LA English

L8 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1999:76842 BIOSIS
DN PREV199900076842
TI Involvement of interleukin-1 in the development of ulcerative **colitis** induced by dextran sulfate sodium in mice.
AU Arai, Yoshinori (1); Takanashi, Hitoshi; Kitagawa, Hiroshi; **Okayasu, Isao**
CS (1) Lab. Pharmacol. Preclin. Dev. Lab., Res. Dev. Div., Nippon Hoechst Marion Roussel Ltd., 1-3-2 Minamidai, Kawagoe 350-11 Japan
SO Cytokine, (Nov., 1998) Vol. 10, No. 11, pp. 890-896.
ISSN: 1043-4666.
DT Article
LA English
AB Dextran sulfate sodium (DSS)-induced **colitis** in mice has been recognized as a model for human ulcerative **colitis**. Using this model, the effects of anti-murine interleukin 1beta (IL-1beta) antibodies (anti-muIL-1beta) and recombinant murine IL-1 receptor type I (rmuIL-1R) on the development of **colitis** were examined to determine whether IL-1 plays a role in **colitis**. Furthermore, RT-PCR amplification was used to examine for the presence of mRNAs for IL-1alpha and IL-1beta

in the large intestine. In mice with **colitis** induced by DSS, administration of anti-muIL-1beta (5 mg/kg, once/week, i.p.) significantly suppressed body weight loss and shortening of the large intestine. Administration of rmuIL-1R (0.2 mg/kg or 1.0 mg/kg, once/day, i.v.) significantly suppressed shortening of the large intestine. Expression of mRNAs for IL-1alpha and IL-1beta was observed in the large intestine of mice which received distilled water containing 3% DSS for 5 days. The expression tended to increase in mice which received DSS for 11 days. In contrast, mRNA expression was not observed in mice which received distilled water without DSS. These results clearly demonstrate that IL-1 is involved in the development of DSS-induced **colitis** in mice and suggest that downregulation of IL-1 might be useful for the treatment of patients with ulcerative **colitis**.

L8 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1998:118814 BIOSIS
DN PREV199800118814
TI Relationship between fecal bile acids and the occurrence of colorectal neoplasia in experimental murine ulcerative **colitis**.
AU Kajiura, Ken; Ohkusa, Toshifumi (1); **Okayasu, Isao**
CS (1) First Dep. Internal Med., Tokyo Med. Dental Univ. Sch. Med., Yushima 1-5, Bunkyo-ku, Tokyo 113 Japan
SO Digestion, (Jan.-Feb., 1998) Vol. 59, No. 1, pp. 69-72.
ISSN: 0012-2823.
DT Article
LA English
AB Objective: The possible role of fecal bile acids in colorectal carcinogenesis in ulcerative **colitis** has been reported. In this study, we investigated the relationship between fecal bile acids and the occurrence of colorectal neoplasia in experimental murine **colitis** induced by dextran sulfate sodium. Methods: Colorectal neoplasia in experimental **colitis** was induced by dextran sulfate sodium subsequent to a single azoxymethane pretreatment. Fecal bile acids were analyzed by gas-liquid chromatography. Results: Multiple high-grade dysplasias (intramucosal adenocarcinoma) and inflammatory changes were seen in all mice administered dextran sulfate sodium and azoxymethane. Inflammatory changes were also observed in all mice given dextran sulfate sodium only, while neither tumor nor inflammatory changes were detected in any of the control mice. Significant increases in cholic acid were observed in the mice of the colorectal tumor and experimental **colitis** groups during the experimental period, while in the control mice, no significant changes in fecal bile acids were observed. Conclusion: It is suggested that fecal cholic acid and **colitis** may be intimately related to the development of colorectal neoplasia in this experimental model of murine **colitis** as well as in ulcerative **colitis**.

L8 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1997:201438 BIOSIS
DN PREV199799500641
TI Distinctive diffuse duodenitis associated with ulcerative **colitis**.
AU Mitomi, Hiroyuki (1); Atari, Eio; Uesugi, Hidenaga; Nishiyama, Yasuhiko; Igarashi, Masahiro; Arai, Nobuyasu; Ihara, Atsushi; **Okayasu, Isao**
CS (1) Dep. Pathol., Sch. Med., Kitasato Univ. East Hosp., 2-1-1 Asamizo-dai, Sagamihara, Kanagawa 228 Japan
SO Digestive Diseases and Sciences, (1997) Vol. 42, No. 3, pp. 684-693.
ISSN: 0163-2116.
DT (CASE STUDY)
LA English

L8 ANSWER 11 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1996:280686 BIOSIS
DN PREV199699003042

TI Population changes in immunoglobulin-containing mononuclear cells in dextran sulfate sodium-induced **colitis**.
AU Tokoi, Shinsuke (1); Ohkusa, Toshifumi; **Okayasu, Isao**; Nakamura, Kyoichi
CS (1) First Dep. Pathol., Tokyo Medical Dental University, 1-5-45 Yushima, Bunkyo-ku, Tokyo 113 Japan
SO Journal of Gastroenterology, (1996) Vol. 31, No. 2, pp. 182-188. ISSN: 1340-9077.
DT Article
LA English
AB To investigate the relation of immunoglobulin containing cells in the colonic mucosa to mucosal inflammation, we immunohistochemically examined the localization of immunoglobulin-containing mononuclear cells in the lamina propria in dextran sulfate sodium induced **colitis** in mice. Mice were treated repeatedly with 3% dextran sulfate sodium (MW 54000) solution or distilled water for a total of 170 days (chronic model), or for 85 days (subacute model) or for 10 days (acute model). IgG, IgA, and IgM-containing mononuclear cells were studied by enzyme immunostaining. The number of IgA- and IgG-containing cells gradually and significantly increased in the acute, subacute, and chronic models, in that order (P lt 0.01 or 0.05). However, the numbers of IgM-containing cells in the three models were similar to that in the controls. These findings resembled those of human ulcerative **colitis**. In this dextran sulfate sodium-induced **colitis**, IgA-containing mononuclear cells may play an essential role in the mucosal immune system is the acute. subacute, and chronic phases. The finding that IgG-containing mononuclear cells increased substantially in the chronic phase suggests that IgG plays an important role in the mucosal inflammatory reaction during the chronic phase.

L8 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1995:218063 BIOSIS
DN PREV199598232363
TI Changes in bacterial phagocytosis of macrophages in experimental ulcerative **colitis**.
AU Ohkusa, Toshifumi (1); **Okayasu, Isao**; Tokoi, Shinsuke; Araki, Akihiro; Ozaki, Yoshinori
CS (1) First Dep. Intern. Med., Tokyo Med. Dental Univ. Sch. Med., Yushima 1-5, Bunkyo-ku, Tokyo 113 Japan
SO Digestion, (1995) Vol. 56, No. 2, pp. 159-164. ISSN: 0012-2823.
DT Article
LA English
AB We previously reported that numerous macrophages which phagocytosed dextran sulfate sodium were observed to have accumulated in the mucosal lesions and in the spleen in experimental ulcerative **colitis** induced in mice with dextran sulfate sodium. In this paper, we investigated the bacterial phagocytic ability of macrophages which were isolated from spleens of mice treated with 3% dextran sulfate sodium for 6 months. In this model, the number of phagocytosed bacteria (Listeria monocytogenes) and the phagocytic index were significantly decreased. The phagocytic ability of splenic macrophages obtained from nontreated mice was also evaluated by incubating with dextran sulfate sodium in vitro, and adding bacteria. The number of phagocytosed bacteria and the phagocytic index were also significantly decreased. These observations suggest that the decrease in bacterial phagocytosis in this model by macrophages that phagocytosed dextran sulfate sodium indicates a decline of the mucosal defense system to bacteria.

L8 ANSWER 13 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1993:304048 BIOSIS
DN PREV199396022273
TI Development of colorectal cancer in ulcerative **colitis**, clinicopathological study of 347 patients and new concepts of cancer

development from analysis of mucosal cell proliferation activity.
AU **Okayasu, Isao (1)**; Fujiwara, Mutsunori; Takemura, Tamiko; Toyoshima, Hiroshi; Nakamura, Kyoichi (1)
CS (1) Dep. Pathol., Sch. Med., Tokyo Medical and Dent. Univ., Tokyo Japan
SO Stomach and Intestine, (1993) Vol. 28, No. 2, pp. 171-179.
ISSN: 0536-2180.
DT Article
LA Japanese
SL Japanese; English
AB By clinicopathological analysis of 347 patients (male 178, female 169) of ulcerative **colitis** (UC) on which follow-up study has been performed at three hospital in Tokyo metropolitan area, cumulative probabilities of developing colorectal cancer were estimated as follows: 10 year period, 1.1%; 15 year period, 3.2%; 20 year period, 11.1%. Histopathological study of 32 surgically resected specimens of the colon and 23 remaining rectums after colectomy showed a high incidence of dysplasia (12.5%, 8.7% respectively). These results suggest that carcinoma and dysplasia may arise in Japanese UC patients with long suffering periods. UC patients with neoplasms were significantly younger (46.8 years of age) and more female dominant than those with sporadic colorectal cancers. Histopathological findings of mitosis index, AgNORs granules and PCNA positive cells revealed that mitosis and cellular proliferation of mucosal epithelium obtained from active UC patients were more prominent than those from UC in remission phase or non-UC patients. In conclusion, the repetition of necrosis and regeneration may cause the pre-malignant change and the increase in incidence of colorectal cancer.

L8 ANSWER 14 OF 15 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1993:253222 BIOSIS
DN PREV199395132397
TI Bacterial invasion into the colonic mucosa in ulcerative **colitis**.
AU Ohkusa, Toshifumi (1); **Okayasu, Isao**; Tokoi, Shinsuke; Ozaki, Yoshinori
CS (1) First Dep. Internal Med., Sch. Med., Tokyo Med. Dental Univ., 1-5 Yushima, Bunkyo-ku, Tokyo 113 Japan
SO Journal of Gastroenterology and Hepatology, (1993) Vol. 8, No. 1, pp. 116-118.
ISSN: 0815-9319.
DT Article
LA English
AB This study investigated interactions between mucosal lesions and bacterial invasion in ulcerative **colitis** using the acridine-orange staining method. In all 16 cases of ulcerative **colitis**, the mucosa was found to be invaded by small rods and cocci. In five of 10 controls, bacteria were seen only adhering to the mucosa and no bacteria were detected in the five remaining cases. It is suggested that the presence of bacteria in the colonic mucosa may be a factor responsible for the persistence or aggravation of ulcerative **colitis**.

L8 ANSWER 15 OF 15 JAPIO COPYRIGHT 2002 JPO
AN 1995-126177 JAPIO
TI THERAPEUTIC AGENT FOR **COLITIS** ULCEROSA
IN FUJIWARA MUTSUNORI; **OKAYASU ISAO**; IWANA HIROKAZU; KANEKO TSUTOMU; TAKETOMO TADAO; SUMIO HAJIME
PA MEIJI MILK PROD CO LTD, JP (CO 000613)
PI JP 07126177 A 19950516 Heisei
AI JP1993-271771 (JP05271771 Heisei) 19931029
SO PATENT ABSTRACTS OF JAPAN (CD-ROM), Unexamined Applications, Vol. 95, No. 5
AB PURPOSE: To provide a therapeutic agent for **colitis** ulcerosa, containing cell bodies of a Lactobacillus bifidus as the active component, excellent in its effect and capable of large volume administration with safety.

CONSTITUTION: This therapeutic agent contains cell bodies of a Lactobacillus bifidus (e.g. Bifidobacterium longum No7 (FERMP-13610)) belonging to Bifidobacterium as the active component. The Lactobacillus bifidus is an enterobacterium of human and animals and very important for maintenance of health condition from old times and this therapeutic agent has various effects, e.g. inhibition of production of harmful substances such as a cancerogenic substance, prevention of intestinal infection with a pathogen, prevention of growth of harmful enterobacteria, synthesis of vitamin B1, B2, B6, B12 and K, promotion of digestion and absorption and immunological enhancement. The dosage is .gtoreq.1.times.106/day per an adult on viable cell base. This therapeutic agent is excellent in safety, the therapeutic effect and prevention of recurrence and can be used in combination with salazopyrin or prednizolone.

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LEGAL REPRESENTATIVE: Sterne, Kessler, Goldstein & Fox
NUMBER OF CLAIMS: 9
EXEMPLARY CLAIM: 5
NUMBER OF DRAWINGS: 2 Drawing Figure(s); 2 Drawing Page(s)
LINE COUNT: 661

=> d bib ab l2 1-8

L2 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
AN 1992:452566 BIOSIS
DN BA94:93966
TI AIR ENEMA REVISITED IN ASSESSMENT OF COLITIS.
AU LINDSTROM E; NOREN B
CS DEP. SURGERY, UNIV. HOSP., S-581 85 LINKOPING, SWED.
SO ACTA RADIOL (CPH), (1992) 33 (4), 360-364.
CODEN: ACRAE3. ISSN: 0284-1851.
FS BA; OLD
LA English
AB Plain radiographs and air enema were performed in 37 patients with ulcerative colitis, 7 patients with proctitis, and 8 patients with Crohn's disease. The air enema was superior to plain radiographs for **diagnosing colitis**, and for delineating the extent of disease and the degree of mucosal involvement. The air enema is simple to perform and easy to evaluate as shown by an almost complete agreement between 2 observers.

L2 ANSWER 2 OF 8 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
AN 92232911 EMBASE
DN 1992232911
TI Air enema revisited in assessment of colitis.
AU Lindstrom E.; Noren B.
CS Department of Surgery, University Hospital,S-581 85 Linkoping, Sweden
SO Acta Radiologica, (1992) 33/4 (360-364).
ISSN: 0248-1851 CODEN: ACRAE3
CY Denmark
DT Journal; Article
FS 014 Radiology
048 Gastroenterology
LA English
SL English
AB Plain radiographs and air enema were performed in 37 patients with ulcerative colitis, 7 patients with proctitis, and 8 patients with Crohn's disease. The air enema was superior to plain radiographs for **diagnosing colitis**, and for delineating the extent of disease and the degree of mucosal involvement. The air enema is simple to perform and easy to evaluate as shown by an almost complete agreement between 2 observers.

L2 ANSWER 3 OF 8 MEDLINE
AN 92338026 MEDLINE
DN 92338026 PubMed ID: 1633048
TI Air enema revisited in assessment of colitis.
AU Lindstrom E; Noren B
CS Department of Surgery, University Hospital, Linkoping, Sweden.
SO ACTA RADIOLOGICA, (1992 Jul) 33 (4) 360-4.
Journal code: ATA; 8706123. ISSN: 0284-1851.
CY Denmark
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199208
ED Entered STN: 19920911
Last Updated on STN: 19920911

Entered Medline: 19920825
AB Plain radiographs and air enema were performed in 37 patients with ulcerative colitis, 7 patients with proctitis, and 8 patients with Crohn's disease. The air enema was superior to plain radiographs for **diagnosing colitis**, and for delineating the extent of disease and the degree of mucosal involvement. The air enema is simple to perform and easy to evaluate as shown by an almost complete agreement between 2 observers.

L2 ANSWER 4 OF 8 MEDLINE
AN 87081305 MEDLINE
DN 87081305 PubMed ID: 3792790
TI **Diagnosing colitis**. Biopsy is best.
AU Surawicz C M
SO GASTROENTEROLOGY, (1987 Feb) 92 (2) 538-40.
Journal code: FH3; 0374630. ISSN: 0016-5085.
CY United States
DT Editorial
LA English
FS Abridged Index Medicus Journals; Priority Journals
EM 198702
ED Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19870217

L2 ANSWER 5 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 2001:499304 SCISEARCH
GA The Genuine Article (R) Number: 429KA
TI **Diagnosing colitis**: A prospective study on essential parameters for reaching an accurate diagnosis.
AU Dejaco C (Reprint); Oesterreicher C; Puespoek A; Birner P; Poetzi R; Gangl A; Oberhuber G
CS Dept Gastroenterol, Vienna, Austria; Dept Pathol, Vienna, Austria
CYA Austria
SO GASTROENTEROLOGY, (APR 2001) Vol. 120, No. 5, Supp. [1], pp. A282-A282. MA 1458.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0016-5085.
DT Conference; Journal
LA English
REC Reference Count: 0

L2 ANSWER 6 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 92:435689 SCISEARCH
GA The Genuine Article (R) Number: JD998
TI AIR ENEMA REVISITED IN ASSESSMENT OF COLITIS
AU LINDSTROM E (Reprint); NOREN B
CS LINKOPING UNIV HOSP, DEPT SURG, S-58185 LINKOPING, SWEDEN (Reprint); LINKOPING UNIV HOSP, DEPT RADIOL, S-58185 LINKOPING, SWEDEN
CYA SWEDEN
SO ACTA RADIOLOGICA, (JUL 1992) Vol. 33, No. 4, pp. 360-364.
ISSN: 0567-8056.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 10
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Plain radiographs and air enema were performed in 37 patients with ulcerative colitis, 7 patients with proctitis, and 8 patients with Crohn's disease. The air enema was superior to plain radiographs for **diagnosing colitis**, and for delineating the extent of disease and the degree of mucosal involvement. The air enema is simple to perform and easy to evaluate as shown by an almost complete agreement

TITLE: **Diagnosing colitis**: A prospective study on essential parameters for reaching an accurate diagnosis.
AUTHOR: Dejaco C (Reprint); Oesterreicher C; Puespoek A; Birner P; Poetzi R; Gangl A; Oberhuber G
CORPORATE SOURCE: Dept Gastroenterol, Vienna, Austria; Dept Pathol, Vienna, Austria
COUNTRY OF AUTHOR: Austria
SOURCE: GASTROENTEROLOGY, (APR 2001) Vol. 120, No. 5, Supp. [1], pp. A282-A282. MA 1458.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0016-5085.
DOCUMENT TYPE: Conference; Journal
LANGUAGE: English
REFERENCE COUNT: 0

L2 ANSWER 6 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 92:435689 SCISEARCH
THE GENUINE ARTICLE: JD998
TITLE: AIR ENEMA REVISITED IN ASSESSMENT OF COLITIS
AUTHOR: LINDSTROM E (Reprint); NOREN B
CORPORATE SOURCE: LINKOPING UNIV HOSP, DEPT SURG, S-58185 LINKOPING, SWEDEN (Reprint); LINKOPING UNIV HOSP, DEPT RADIOL, S-58185 LINKOPING, SWEDEN
COUNTRY OF AUTHOR: SWEDEN
SOURCE: ACTA RADIOLOGICA, (JUL 1992) Vol. 33, No. 4, pp. 360-364.
ISSN: 0567-8056.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 10
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

L2 ANSWER 7 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 87:45923 SCISEARCH
THE GENUINE ARTICLE: F6575
TITLE: **DIAGNOSING COLITIS** - BIOPSY IS BEST
AUTHOR: SURAWICZ C M (Reprint)
CORPORATE SOURCE: UNIV WASHINGTON, SCH MED, SEATTLE, WA, 98195
COUNTRY OF AUTHOR: USA
SOURCE: GASTROENTEROLOGY, (1987) Vol. 92, No. 2, pp. 538-540.
DOCUMENT TYPE: Editorial; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: ENGLISH
REFERENCE COUNT: 8

L2 ANSWER 8 OF 8 USPATFULL
ACCESSION NUMBER: 93:61012 USPATFULL
TITLE: Monoclonal antibodies specific for Toxin B of Clostridium difficile
INVENTOR(S): Coughlin, Richard T., Leicester, MA, United States
Marciani, Dante J., Hopkinton, MA, United States
PATENT ASSIGNEE(S): Cambridge Bioscience Corporation, Worcester, MA, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5231003		19930727
APPLICATION INFO.:	US 1990-522881		19900511 (7)

DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Ceperley, Mary E.
ASSISTANT EXAMINER: Bidwell, Carol E.

2 ANSWER 1 OF 8 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1992:452566 BIOSIS
DOCUMENT NUMBER: BA94:93966
TITLE: AIR ENEMA REVISITED IN ASSESSMENT OF COLITIS.
AUTHOR(S): LINDSTROM E; NOREN B
CORPORATE SOURCE: DEP. SURGERY, UNIV. HOSP., S-581 85 LINKOPING, SWED.
SOURCE: ACTA RADIOL (CPH), (1992) 33 (4), 360-364.
CODEN: ACRAE3. ISSN: 0284-1851.
FILE SEGMENT: BA; OLD
LANGUAGE: English

L2 ANSWER 2 OF 8 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 92232911 EMBASE
DOCUMENT NUMBER: 1992232911
TITLE: Air enema revisited in assessment of colitis.
AUTHOR: Lindstrom E.; Noren B.
CORPORATE SOURCE: Department of Surgery, University Hospital,S-581 85 Linkoping, Sweden
SOURCE: Acta Radiologica, (1992) 33/4 (360-364).
ISSN: 0248-1851 CODEN: ACRAE3
COUNTRY: Denmark
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 014 Radiology
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

L2 ANSWER 3 OF 8 MEDLINE
ACCESSION NUMBER: 92338026 MEDLINE
DOCUMENT NUMBER: 92338026 PubMed ID: 1633048
TITLE: Air enema revisited in assessment of colitis.
AUTHOR: Lindstrom E; Noren B
CORPORATE SOURCE: Department of Surgery, University Hospital, Linkoping, Sweden.
SOURCE: ACTA RADIOLOGICA, (1992 Jul) 33 (4) 360-4.
Journal code: ATA; 8706123. ISSN: 0284-1851.
PUB. COUNTRY: Denmark
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199208
ENTRY DATE: Entered STN: 19920911
Last Updated on STN: 19920911
Entered Medline: 19920825

L2 ANSWER 4 OF 8 MEDLINE
ACCESSION NUMBER: 87081305 MEDLINE
DOCUMENT NUMBER: 87081305 PubMed ID: 3792790
TITLE: **Diagnosing colitis**. Biopsy is best.
AUTHOR: Surawicz C M
SOURCE: GASTROENTEROLOGY, (1987 Feb) 92 (2) 538-40.
Journal code: FH3; 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
Editorial
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198702
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19870217

L2 ANSWER 5 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
ACCESSION NUMBER: 2001:499304 SCISEARCH
THE GENUINE ARTICLE: 429KA

between 2 observers.

L2 ANSWER 7 OF 8 SCISEARCH COPYRIGHT 2002 ISI (R)
AN 87:45923 SCISEARCH
GA The Genuine Article (R) Number: F6575
TI **DIAGNOSING COLITIS** - BIOPSY IS BEST
AU SURAWICZ C M (Reprint)
CS UNIV WASHINGTON, SCH MED, SEATTLE, WA, 98195
CYA USA
SO GASTROENTEROLOGY, (1987) Vol. 92, No. 2, pp. 538-540.
DT Editorial; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 8

L2 ANSWER 8 OF 8 USPATFULL
AN 93:61012 USPATFULL
TI Monoclonal antibodies specific for Toxin B of Clostridium difficile
IN Coughlin, Richard T., Leicester, MA, United States
Marciani, Dante J., Hopkinton, MA, United States
PA Cambridge Bioscience Corporation, Worcester, MA, United States (U.S. corporation)
PI US 5231003 19930727
AI US 1990-522881 19900511 (7)
DT Utility
FS Granted
EXNAM Primary Examiner: Ceperley, Mary E.; Assistant Examiner: Bidwell, Carol E.
LREP Sterne, Kessler, Goldstein & Fox
CLMN Number of Claims: 9
ECL Exemplary Claim: 5
DRWN 2 Drawing Figure(s); 2 Drawing Page(s)
LN.CNT 661
AB Monoclonal antibodies specific for Toxin B of Clostridium difficile are provided. Further, methods for making and using the antibodies are given, particularly the use of the antibodies for the detection of C. difficile.

L3 ANSWER 1 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1998:425849 BIOSIS
DOCUMENT NUMBER: PREV199800425849
TITLE: Impact of circulatory analysis by computer-assisted endoscopy.
AUTHOR(S): Tsuji, Shingo (1); Kawano, Sunao (1); Tsujii, Masahiko (1); **Sato, Nobuhiro**; Hori, Masatsugu (1)
CORPORATE SOURCE: (1) First Dep. Med., Osaka Univ. Sch. Med., 2-2 Yamadaoka, Suita 565-0871, Osaka Japan
SOURCE: Lemke, H. U. [Editor]; Vannier, M. W. [Editor]; Inamura, K. [Editor]; Farman, A. G. [Editor]. International Congress Series, (1998) No. 1165, pp. 874. International Congress Series; CAR '98: Computer assisted radiology and surgery. Publisher: Elsevier Science Publishers B.V. PO Box 211, Sara Burgerhartstraat 25, 1000 AE Amsterdam, The Netherlands.
Meeting Info.: 12th International Symposium and exhibition Tokyo, Japan June 24-27, 1998
ISSN: 0531-5131. ISBN: 0-444-82973-3.
DOCUMENT TYPE: Book; Conference
LANGUAGE: English

L3 ANSWER 2 OF 2 BIOSIS COPYRIGHT 2002 BIOLOGICAL ABSTRACTS INC.
ACCESSION NUMBER: 1995:358507 BIOSIS
DOCUMENT NUMBER: PREV199598372807
TITLE: Colonic mucosal hemodynamics and tissue oxygenation in patients with ulcerative **colitis**: Investigation by organ reflectance spectrophotometry.
AUTHOR(S): Tsujii, Masahiko; Kawano, Sunao; Tsuji, Shingo; Kobayashi, Ichizo; Takei, Yoshiyuki; Nagano, Kouichi; Fusamoto, Hideyuki; Kamada, Takenobu; Ogihara, Tatsuo; **Sato, Nobuhiro**
CORPORATE SOURCE: First Dep. Med., Osaka Univ. Sch. Med., 2-2 Yamada-oka, Suita 565 Japan
SOURCE: Journal of Gastroenterology, (1995) Vol. 30, No. 2, pp. 183-188.
DOCUMENT TYPE: Article
LANGUAGE: English

=>

5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:83011 CAPLUS
DOCUMENT NUMBER: 130:323482
TITLE: Inflammatory bowel disease and intestinal bacteria
AUTHOR(S): **Okusa, Toshifumi**
CORPORATE SOURCE: The First Department of Internal Medicine, Tokyo Medical and Dental University, Japan
SOURCE: Rinsho Kagaku (Osaka) (1998), 34(12), 1593-1597
CODEN: RIKAER; ISSN: 0385-0323
PUBLISHER: Esuato K. K.
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese

=> d 15 ab

L5 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2002 ACS
AB A review with 30 refs. describes the pathogenic microorganism of ulcerative colitis and Crohn's disease, and inflammatory bowl disease-causing structural components and products (endotoxin, heat shock protein, and other inflammatory substances) of bacteria.

ANSWER 1 OF 15 CABA COPYRIGHT 2002 CABI

ACCESSION NUMBER: 1998:169113 CABA

DOCUMENT NUMBER: 981415717

TITLE: Role of short-chain fatty acids in the hind gut

AUTHOR: Engelhardt, W. von; Bartels, J.; Kirschberger, S.; Duttingdorf, H. D. M. zu; Busche, R.; Von Engelhardt, W.; Zu Duttingdorf, H. D. M.

CORPORATE SOURCE: Department of Physiology and Department of Biochemistry, School of Veterinary Medicine, Hannover, Germany.

SOURCE: Veterinary Quarterly, (1998) Vol. 20, No. S3, pp. S52-S59. 105 ref.
Meeting Info.: Conference proceedings, Gastrointestinal disorders in juveniles, Lelystad, Netherlands, 16-17 September, 1997.
ISSN: 0165-2176

DOCUMENT TYPE: Conference Article; Journal

LANGUAGE: English

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=> File medline
FILE 'MEDLINE' ENTERED AT 11:51:58 ON 18 JAN 2002

 FILE LAST UPDATED: 2 JAN 2002 (20020102/UP). FILE COVERS 1958 TO DATE.

 On April 22, 2001, MEDLINE was reloaded. See HELP RLOAD for details.

 MEDLINE now contains IN-PROCESS records. See HELP CONTENT for details.

 MEDLINE is now updated 4 times per week. A new current-awareness alert
 frequency (EVERYUPDATE) is available. See HELP UPDATE for more information.

 MEDLINE thesauri in the /CN, /CT, and /MN fields incorporate the
 MeSH 2001 vocabulary. Enter HELP THESAURUS for details.

 The OLDMEDLINE file segment now contains data from 1958 through 1965.
 Enter HELP CONTENT for details.

 Left, right, and simultaneous left and right truncation are available in the
 Basic Index. See HELP SFIELDS for details.

 THIS FILE CONTAINS CAS REGISTRY NUMBERS FOR EASY AND ACCURATE
 SUBSTANCE IDENTIFICATION.

=>
=> d que L5

L1   (      1768) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L2   (       559) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L3   (      1938) SEA FILE=MEDLINE ABB=ON  L1 OR L2
L4   (     14863) SEA FILE=MEDLINE ABB=ON  COLITIS, ULCERATIVE/CT
L5             1 SEA FILE=MEDLINE ABB=ON  L3 AND L4


=> d que L10

L6   (      1768) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L7   (       559) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L8   (      1938) SEA FILE=MEDLINE ABB=ON  L6 OR L7
L9   (      1513) SEA FILE=MEDLINE ABB=ON  MODELS, ANIMAL/CT
L10            2 SEA FILE=MEDLINE ABB=ON  L8 AND L9


=> d que L13

L11  (     14863) SEA FILE=MEDLINE ABB=ON  COLITIS, ULCERATIVE/CT
L12  (      1513) SEA FILE=MEDLINE ABB=ON  MODELS, ANIMAL/CT
L13            2 SEA FILE=MEDLINE ABB=ON  L11 AND L12


=> d que L19

L14  (      1768) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L15  (       559) SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L16  (      1938) SEA FILE=MEDLINE ABB=ON  L14 OR L15
L17  (     11179) SEA FILE=MEDLINE ABB=ON  VACCINES, BACTERIAL/CT
L18  (        19) SEA FILE=MEDLINE ABB=ON  L16 AND L17
L19            10 SEA FILE=MEDLINE ABB=ON  L18 AND FUSOBACTERI##/TI
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```
=> d que L26

L20 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L21 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L22 (      1938)SEA FILE=MEDLINE ABB=ON  L20 OR L21
L23 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L24 (    401613)SEA FILE=MEDLINE ABB=ON  EVALUATION STUDIES+NT/CT
L25 (       168)SEA FILE=MEDLINE ABB=ON  L22 AND L24
L26           2 SEA FILE=MEDLINE ABB=ON  L25 AND L23


=> d que L31

L27 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L28 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L29 (      1938)SEA FILE=MEDLINE ABB=ON  L27 OR L28
L30 (     53714)SEA FILE=MEDLINE ABB=ON  DRUG EVALUATION/CT OR DRUG EVALUATION,
                 PRECLINICAL/CT
L31           4 SEA FILE=MEDLINE ABB=ON  L29 AND L30


=>

=> d que L38

L32 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L33 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L34 (      1938)SEA FILE=MEDLINE ABB=ON  L32 OR L33
L35 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L36 (     24202)SEA FILE=MEDLINE ABB=ON  ANTIGENS, BACTERIAL/CT
L37 (        63)SEA FILE=MEDLINE ABB=ON  L34 AND L36
L38           1 SEA FILE=MEDLINE ABB=ON  L37 AND L35


=> d que L45

L39 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L40 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L41 (      1938)SEA FILE=MEDLINE ABB=ON  L39 OR L40
L42 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L43 (       836)SEA FILE=MEDLINE ABB=ON  ADHESINS, BACTERIAL/CT
L44 (         5)SEA FILE=MEDLINE ABB=ON  L41 AND L43
L45           0 SEA FILE=MEDLINE ABB=ON  L44 AND L42


=> d que L51

L46 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L47 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L48 (      1938)SEA FILE=MEDLINE ABB=ON  L46 OR L47
L49 (     32390)SEA FILE=MEDLINE ABB=ON  LIPOPOLYSACCHARIDES+NT/CT OR PEPTIDOGL
                 YCAN/CT
L50 (       101)SEA FILE=MEDLINE ABB=ON  L48 AND L49
L51           1 SEA FILE=MEDLINE ABB=ON  L50 AND VARIUM/TI


=> d que L58

L52 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L53 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
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L54 (      1938)SEA FILE=MEDLINE ABB=ON  L52 OR L53
L55 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L56 (     29136)SEA FILE=MEDLINE ABB=ON  BACTERIAL TOXINS+NT/CT
L57 (        29)SEA FILE=MEDLINE ABB=ON  L54 AND L56
L58            0 SEA FILE=MEDLINE ABB=ON  L57 AND L55


=> d que L65

L59 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L60 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L61 (      1938)SEA FILE=MEDLINE ABB=ON  L59 OR L60
L62 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L63 (     42654)SEA FILE=MEDLINE ABB=ON  ENDOTOXINS+NT/CT
L64 (       121)SEA FILE=MEDLINE ABB=ON  L61 AND L63
L65            2 SEA FILE=MEDLINE ABB=ON  L64 AND L62


=> d que L72

L66 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L67 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L68 (      1938)SEA FILE=MEDLINE ABB=ON  L66 OR L67
L69 (        69)SEA FILE=MEDLINE ABB=ON  VARIUM
L70 (     28481)SEA FILE=MEDLINE ABB=ON  ANTIBODIES, BACTERIAL+NT/CT
L71 (        94)SEA FILE=MEDLINE ABB=ON  L68 AND L70
L72            1 SEA FILE=MEDLINE ABB=ON  L71 AND L69


=> d que L78

L73 (     14863)SEA FILE=MEDLINE ABB=ON  COLITIS, ULCERATIVE/CT
L74 (      1587)SEA FILE=MEDLINE ABB=ON  BUTYRIC ACID/CT
L75 (        26)SEA FILE=MEDLINE ABB=ON  L73 AND L74
L76 (     37918)SEA FILE=MEDLINE ABB=ON  INTESTINAL MUCOSA/CT
L77 (        15)SEA FILE=MEDLINE ABB=ON  L75 AND L76
L78           10 SEA FILE=MEDLINE ABB=ON  L77 AND COLITIS/TI


=> d que L83

L79 (      1768)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM+NT,OLD/CT
L80 (       559)SEA FILE=MEDLINE ABB=ON  FUSOBACTERIUM INFECTIONS/CT
L81 (      1938)SEA FILE=MEDLINE ABB=ON  L79 OR L80
L82 (      6636)SEA FILE=MEDLINE ABB=ON  VACCINES, INACTIVATED/CT OR VACCINES,
                   ATTENUATED/CT
L83            1 SEA FILE=MEDLINE ABB=ON  L81 AND L82


=>

=> s l5 or l10 or l13 or l19 or l26 or l31 or l38 or l45 or l51 or l58 or l65 or
l72 or l78 or l83

L240          35 L5 OR L10 OR L13 OR L19 OR L26 OR L31 OR L38 OR L45 OR L51 OR
                 L58 OR L65 OR L72 OR L78 OR L83

=>

=> File CABA
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FILE 'CABA' ENTERED AT 11:52:21 ON 18 JAN 2002
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FILE COVERS 1973 TO 11 Jan 2002 (20020111/ED)

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=>

=> d que L88

L84 (         13)SEA FILE=CABA ABB=ON  (F OR FUSOBACTERIUM) (W) VARIUM
L85 (          2)SEA FILE=CABA ABB=ON  FUSOBACTERIAL
L86 (         15)SEA FILE=CABA ABB=ON  L84 OR L85
L87 (        644)SEA FILE=CABA ABB=ON  ULCERATIVE COLITIS
L88            0 SEA FILE=CABA ABB=ON  L86 AND L87


=> d que L94

L89 (         13)SEA FILE=CABA ABB=ON  (F OR FUSOBACTERIUM) (W) VARIUM
L90 (          2)SEA FILE=CABA ABB=ON  FUSOBACTERIAL
L91 (         15)SEA FILE=CABA ABB=ON  L89 OR L90
L92 (        644)SEA FILE=CABA ABB=ON  ULCERATIVE COLITIS
L93 (          0)SEA FILE=CABA ABB=ON  L91 AND L92
L94            0 SEA FILE=CABA ABB=ON  L91 AND L93


=> d que L99

L95 (         13)SEA FILE=CABA ABB=ON  (F OR FUSOBACTERIUM) (W) VARIUM
L96 (          2)SEA FILE=CABA ABB=ON  FUSOBACTERIAL
L97 (         15)SEA FILE=CABA ABB=ON  L95 OR L96
L98 (        282)SEA FILE=CABA ABB=ON  FUSOBACTERIUM/TI OR FUSOBACTERIAL/TI
L99            8 SEA FILE=CABA ABB=ON  L97 AND L98


=>

=> s l88 or l94 or l99

L241           8 L88 OR L94 OR L99

=>

=> File caplus

FILE 'CAPLUS' ENTERED AT 11:52:28 ON 18 JAN 2002
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26, 1996), unless otherwise indicated in the original publications.
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FILE COVERS 1907 - 18 Jan 2002 VOL 136 ISS 3
FILE LAST UPDATED: 16 Jan 2002 (20020116/ED)

This file contains CAS Registry Numbers for easy and accurate substance identification.

This file supports REG1stRY for direct browsing and searching of all substance data from the REGISTRY file. Enter HELP FIRST for more information.

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CAS roles have been modified effective December 16, 2001. Please check your SDI profiles to see if they need to be revised. For information on CAS roles, enter HELP ROLES at an arrow prompt or use the CAS Roles thesaurus (/RL field) in this file.

The CA Lexicon is now available in the Controlled Term (/CT) field. Enter HELP LEXICON for full details.

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=>

=> d que L102

```
L100(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L101(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
               ULCERATIVE/OBI
L102          0 SEA FILE=CAPLUS ABB=ON  L100 AND L101
```

=> d que L105

```
L103(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L104(      2503)SEA FILE=CAPLUS ABB=ON  "INFECTION (L) BACTERIAL"/CT
L105          1 SEA FILE=CAPLUS ABB=ON  L103 AND L104
```

=> d que L108

```
L106(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L107(     27232)SEA FILE=CAPLUS ABB=ON  VACCINES/CT OR VACCINATE/OBI OR
               VACCINATION/OBI
L108          0 SEA FILE=CAPLUS ABB=ON  L106 AND L107
```

=> d que L111

```
L109(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L110(      3870)SEA FILE=CAPLUS ABB=ON  IMMUNIZATION/CT OR IMMUNIZE/OBI
L111          0 SEA FILE=CAPLUS ABB=ON  L109 AND L110
```

```
=> d que L114

L112(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L113(      9617)SEA FILE=CAPLUS ABB=ON  "VIRULENCE (MICROBIAL)"+OLD/CT
L114          0 SEA FILE=CAPLUS ABB=ON  L112 AND L113


=> d que L117

L115(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L116(      6219)SEA FILE=CAPLUS ABB=ON  IMMUNOTHERAPY+NT,OLD/CT
L117          0 SEA FILE=CAPLUS ABB=ON  L115 AND L116


=> d que L120

L118(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
               ULCERATIVE/OBI
L119(      9617)SEA FILE=CAPLUS ABB=ON  "VIRULENCE (MICROBIAL)"+OLD/CT
L120          1 SEA FILE=CAPLUS ABB=ON  L118 AND L119


=> d que L124

L121(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
               ULCERATIVE/OBI
L122(      3870)SEA FILE=CAPLUS ABB=ON  IMMUNIZATION/CT OR IMMUNIZE/OBI
L123(         2)SEA FILE=CAPLUS ABB=ON  L121 AND L122
L124          1 SEA FILE=CAPLUS ABB=ON  L123 NOT (EPSTEIN-BARR VIRUS)/TI


=> d que L127

L125(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L126(     20528)SEA FILE=CAPLUS ABB=ON  BUTYRATE OR 461-55-2/RN
L127          1 SEA FILE=CAPLUS ABB=ON  L125 AND L126


=> d que L128

L128          7 SEA FILE=CAPLUS ABB=ON  (BUTYRATE AND ULCERATIVE COLITIS)/TI


=> d que L131

L129(        55)SEA FILE=CAPLUS ABB=ON  FUSOBACTERIUM VARIUM+OLD/CT
L130(     30822)SEA FILE=CAPLUS ABB=ON  BUTYRIC OR BUTANOIC OR 107-92-6/RN
L131          5 SEA FILE=CAPLUS ABB=ON  L129 AND L130


=> d que L137

L132(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
               ULCERATIVE/OBI
L133(     27232)SEA FILE=CAPLUS ABB=ON  VACCINES/CT OR VACCINATE/OBI OR
               VACCINATION/OBI
L134(        18)SEA FILE=CAPLUS ABB=ON  L132 AND L133
L135(       820)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/TI
L136(         3)SEA FILE=CAPLUS ABB=ON  L134 AND L135
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L137          2 SEA FILE=CAPLUS ABB=ON  L136 NOT COLECTOMIZED/TI


=> d que L142

L138(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
                ULCERATIVE/OBI
L139(     60986)SEA FILE=CAPLUS ABB=ON  ("ANTIBACTERIAL AGENTS"/CT OR BACTERICI
                DES/CT OR "BACTERICIDES, DISINFECTANTS AND ANTISEPTICS"/CT OR
                "BACTERICIDES, DISINFECTANTS, AND ANTISEPTICS"/CT OR "BACTERICI
                DAL ACTION"/CT OR "BACTERICIDAL ACTION AND BACTERIOSTATIC
                ACTION"/CT OR "BACTERICIDES, DISINFECTANTS, AND ANTISEPTICS
                (L) FUMIGANTS"/CT OR "BACTERICIDES, DISINFECTANTS, AND
                ANTISEPTICS (L) INDUSTRIAL"/CT OR "BACTERICIDES, DISINFECTANTS,
                 AND ANTISEPTICS (L) INDUSTRIAL, SYNERGISTIC"/CT OR "BACTERICID
                ES, DISINFECTANTS, AND ANTISEPTICS (L) SUSTAINED-RELEASE"/CT
                OR "BACTERICIDES, DISINFECTANTS, AND ANTISEPTICS (L) SYNERGISTI
                C"/CT OR "CEPHALOSPORINS (NON-CA HEADING)"/CT OR IODOPHORS/CT
                OR "MICROBICIDAL AND MICROBIOSTATIC ACTION (L) BACTERIOSTATIC"/
                CT)
L140(        25)SEA FILE=CAPLUS ABB=ON  L138 AND L139
L141(       820)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/TI
L142          2 SEA FILE=CAPLUS ABB=ON  L140 AND L141


=> d que L147

L143(      2269)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/OBI OR COLITIS,
                ULCERATIVE/OBI
L144(      8319)SEA FILE=CAPLUS ABB=ON  DISEASE MODELS+NT/CT
L145(        43)SEA FILE=CAPLUS ABB=ON  L143 AND L144
L146(       820)SEA FILE=CAPLUS ABB=ON  ULCERATIVE COLITIS/TI
L147         10 SEA FILE=CAPLUS ABB=ON  L145 AND L146


=>

=> s l102 or l105 or l108 or l111 or l114 or l117 or l120 or l124 or l127 or l128
or l131 or l137 or l142 or l147

L242         29 L102 OR L105 OR L108 OR L111 OR L114 OR L117 OR L120 OR L124 OR
                L127 OR L128 OR L131 OR L137 OR L142 OR L147

=>

=> file Biosis

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CAS REGISTRY NUMBERS AND CHEMICAL NAMES (CNs) PRESENT
FROM JANUARY 1969 TO DATE.

RECORDS LAST ADDED: 16 January 2002 (20020116/ED)

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=>
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=> d que L150

L148(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L149(     13705)SEA FILE=BIOSIS ABB=ON  ULCERATIVE COLITIS
L150          0 SEA FILE=BIOSIS ABB=ON  L148 AND L149


=> d que L153

L151(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L152(     22936)SEA FILE=BIOSIS ABB=ON  COLITIS
L153          0 SEA FILE=BIOSIS ABB=ON  L151 AND L152


=> d que L157

L154(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L155(   1555120)SEA FILE=BIOSIS ABB=ON  DRUG
L156(        29)SEA FILE=BIOSIS ABB=ON  L154 AND L155
L157          6 SEA FILE=BIOSIS ABB=ON  L156 AND FUSOBACTERIUM/TI


=> d que L161

L158(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L159(    132762)SEA FILE=BIOSIS ABB=ON  ANTIBACTERIAL OR ANTIINFECT?
L160(        28)SEA FILE=BIOSIS ABB=ON  L158 AND L159
L161          5 SEA FILE=BIOSIS ABB=ON  L160 AND FUSOBACTERIUM/TI


=> d que L164

L162(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L163(    708880)SEA FILE=BIOSIS ABB=ON  ANTIBOD### OR ANTIGEN#
L164          3 SEA FILE=BIOSIS ABB=ON  L162 AND L163


=> d que L168

L165(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L166(    177476)SEA FILE=BIOSIS ABB=ON  ENDOTOXIN# OR TOXIN# OR LIPOPOLYSACCHAR
                IDE# OR PEPTIDOGLYCAN#
L167(         9)SEA FILE=BIOSIS ABB=ON  L165 AND L166
L168          6 SEA FILE=BIOSIS ABB=ON  L167 AND FUSOBACTERIUM/TI


=> d que L172

L169(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L170(    950809)SEA FILE=BIOSIS ABB=ON  INFECT? OR INVAD? OR INVAS?
L171(        44)SEA FILE=BIOSIS ABB=ON  L170 AND L169
L172          9 SEA FILE=BIOSIS ABB=ON  L171 AND FUSOBACTERIUM/TI


=> d que L176

L173(       131)SEA FILE=BIOSIS ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L174(    765971)SEA FILE=BIOSIS ABB=ON  MODEL#
L175(         7)SEA FILE=BIOSIS ABB=ON  L173 AND L174
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L176          4 SEA FILE=BIOSIS ABB=ON  L175 NOT (COMPUTER SIMULATIONS OR
                GINGIVAL/TI OR COLI/TI)


=> d que L179

L177(    950809)SEA FILE=BIOSIS ABB=ON  INFECT? OR INVAD? OR INVAS?
L178(        64)SEA FILE=BIOSIS ABB=ON  (ULCERATIVE COLITIS/TI) AND (MODEL#/TI)

L179          3 SEA FILE=BIOSIS ABB=ON  L178 AND L177


=> d que L182

L180(    177476)SEA FILE=BIOSIS ABB=ON  ENDOTOXIN# OR TOXIN# OR LIPOPOLYSACCHAR
                IDE# OR PEPTIDOGLYCAN#
L181(        64)SEA FILE=BIOSIS ABB=ON  (ULCERATIVE COLITIS/TI) AND (MODEL#/TI)

L182          3 SEA FILE=BIOSIS ABB=ON  L181 AND L180


=>

=> s l150 or l153 or l157 or l161 or l164 or l168 or l172 or l176 or l179 or l182

L243         28 L150 OR L153 OR L157 OR L161 OR L164 OR L168 OR L172 OR L176 OR
                L179 OR L182

=>

=> File LifeSci

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FILE COVERS 1978 TO 15 Jan 2002 (20020115/ED)

=>

=> d que L186

L183(        42)SEA FILE=LIFESCI ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L184(       691)SEA FILE=LIFESCI ABB=ON  ULCERATIVE COLITIS
L185(        55)SEA FILE=LIFESCI ABB=ON  L183 OR FUSOBACTERIAL
L186          0 SEA FILE=LIFESCI ABB=ON  L185 AND L184


=> d que L190

L187(        42)SEA FILE=LIFESCI ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L188(     37746)SEA FILE=LIFESCI ABB=ON  VACCIN?
L189(        55)SEA FILE=LIFESCI ABB=ON  L187 OR FUSOBACTERIAL
L190          1 SEA FILE=LIFESCI ABB=ON  L189 AND L188


=> d que L196

L191(        42)SEA FILE=LIFESCI ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L192(     24858)SEA FILE=LIFESCI ABB=ON  ANTIINFECTIVE# OR ANTIBACTERIAL# OR
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                ANTIMICROBIAL#
L193(         55)SEA FILE=LIFESCI ABB=ON  L191 OR FUSOBACTERIAL
L194(         10)SEA FILE=LIFESCI ABB=ON  L193 AND L192
L195(        345)SEA FILE=LIFESCI ABB=ON  FUSOBACTERI##/TI
L196           3 SEA FILE=LIFESCI ABB=ON  L194 AND L195


=> d que L200

L197(         42)SEA FILE=LIFESCI ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L198(         55)SEA FILE=LIFESCI ABB=ON  L197 OR FUSOBACTERIAL
L199(      42808)SEA FILE=LIFESCI ABB=ON  SCREEN?
L200           0 SEA FILE=LIFESCI ABB=ON  L198 AND L199


=> d que L204

L201(         42)SEA FILE=LIFESCI ABB=ON  (FUSOBACTERIUM OR F) (W) VARIUM
L202(         55)SEA FILE=LIFESCI ABB=ON  L201 OR FUSOBACTERIAL
L203(     233890)SEA FILE=LIFESCI ABB=ON  ANTIBOD### OR ANTIGEN#
L204           1 SEA FILE=LIFESCI ABB=ON  L202 AND L203


=>

=> s l186 or l190 or L196 or l200 or l204

L244           5 L186 OR L190 OR L196 OR L200 OR L204

=>

=> File Embase

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=>

=> d que L207

L205(         28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L206(      14440)SEA FILE=EMBASE ABB=ON  ULCERATIVE COLITIS
L207           0 SEA FILE=EMBASE ABB=ON  L205 AND L206


=> d que L210

L208(         28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L209(     208338)SEA FILE=EMBASE ABB=ON  BACTERIAL INFECTION+NT/CT
L210           5 SEA FILE=EMBASE ABB=ON  L208 AND L209


=> d que L213
```

```
L211(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L212(     12501)SEA FILE=EMBASE ABB=ON  INTESTINE INFECTION+NT/CT
L213          0 SEA FILE=EMBASE ABB=ON  L211 AND L212


=> d que L216

L214(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L215(     28068)SEA FILE=EMBASE ABB=ON  BACTERIAL VACCINE+NT/CT OR INACTIVATED
                VACCINE+NT/CT
L216          0 SEA FILE=EMBASE ABB=ON  L214 AND L215


=> d que L219

L217(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L218(     67853)SEA FILE=EMBASE ABB=ON  VACCINE+ALL/CT
L219          0 SEA FILE=EMBASE ABB=ON  L217 AND L218


=> d que L222

L220(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L221(      6650)SEA FILE=EMBASE ABB=ON  ADHESIN+NT/CT OR BACTERIUM ADHERENCE+NT
                /CT
L222          0 SEA FILE=EMBASE ABB=ON  L220 AND L221


=> d que L225

L223(     14440)SEA FILE=EMBASE ABB=ON  ULCERATIVE COLITIS
L224(      6650)SEA FILE=EMBASE ABB=ON  ADHESIN+NT/CT OR BACTERIUM ADHERENCE+NT
                /CT
L225          5 SEA FILE=EMBASE ABB=ON  L223 AND L224


=> d que L228

L226(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L227(    310674)SEA FILE=EMBASE ABB=ON  EXPERIMENTAL MODEL/CT OR BIOLOGICAL
                MODEL+NT/CT OR DISEASE MODEL/CT
L228          4 SEA FILE=EMBASE ABB=ON  L227 AND L226


=> d que L234

L229(     14440)SEA FILE=EMBASE ABB=ON  ULCERATIVE COLITIS
L230(      9732)SEA FILE=EMBASE ABB=ON  BUTYRATE OR BUTYRIC
L231(    310674)SEA FILE=EMBASE ABB=ON  EXPERIMENTAL MODEL/CT OR BIOLOGICAL
                MODEL+NT/CT OR DISEASE MODEL/CT
L232(       387)SEA FILE=EMBASE ABB=ON  L231 AND L229
L233(        15)SEA FILE=EMBASE ABB=ON  L232 AND L230
L234         10 SEA FILE=EMBASE ABB=ON  L233 AND COLITIS/TI


=> d que L239

L237(        28)SEA FILE=EMBASE ABB=ON  FUSOBACTERIUM VARIUM/CT
L238(     59335)SEA FILE=EMBASE ABB=ON  DRUG SCREENING+NT/CT
```

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L239          1 SEA FILE=EMBASE ABB=ON  L237 AND L238


=>

=> s l207 or l210 or l213 or l216 or l219 or l222 or l225 or l228 or l234 or l239

L245         23 L207 OR L210 OR L213 OR L216 OR L219 OR L222 OR L225 OR L228 OR
                L234 OR L239

=>

=>

=> dup rem l240 l241 l242 l243 l244 l245
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PROCESSING COMPLETED FOR L241
PROCESSING COMPLETED FOR L242
PROCESSING COMPLETED FOR L243
PROCESSING COMPLETED FOR L244
PROCESSING COMPLETED FOR L245
L246         120 DUP REM L240 L241 L242 L243 L244 L245 (8 DUPLICATES REMOVED)

=>
=> file medline caba caplus biosis lifesci embase

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=>

=> d ibib ab l246 1-120

L246 ANSWER 1 OF 120 MEDLINE
ACCESSION NUMBER: 2001310675 MEDLINE
DOCUMENT NUMBER: 21276893 PubMed ID: 11383596
TITLE: Is infliximab effective for induction of remission in patients with ulcerative colitis?.
COMMENT: Comment on: Inflamm Bowel Dis. 2001 May;7(2):83-8
AUTHOR: Lichtenstein G R
CORPORATE SOURCE: Department of Medicine, Hospital of the University of Pennsylvania, University of Pennsylvania School of Medicine, Philadelphia 19104-4283, USA.. grl@mail.med.upenn.edu
SOURCE: INFLAMMATORY BOWEL DISEASES, (2001 May) 7 (2) 89-93. Journal code: C2I; 9508162. ISSN: 1078-0998.
PUB. COUNTRY: United States
Commentary
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20011022
Last Updated on STN: 20011022
Entered Medline: 20011018

L246 ANSWER 2 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:712146 CAPLUS
DOCUMENT NUMBER: 136:18707
TITLE: Molecular genetics of **ulcerative colitis**-associated colon cancer in the interleukin 2- and .beta.2-microglobulin-deficient mouse
AUTHOR(S): Sohn, Kyoung-Jin; Shah, Samir A.; Reid, Sarah; Choi, Monica; Carrier, Julie; Comiskey, Martina; Terhorst, Cox; Kim, Young-In
CORPORATE SOURCE: Departments of Medicine, University of Toronto, Toronto, ON, M5S 1A8, Can.
SOURCE: Cancer Research (2001), 61(18), 6912-6917
CODEN: CNREA8; ISSN: 0008-5472
PUBLISHER: American Association for Cancer Research
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Mice deficient in .beta.2-microglobulin and interleukin 2 (.beta.2mnull .times. IL-2null) spontaneously develop colon cancer in the setting of chronic ulcerative colitis (UC). We investigated mutations of the Apc and p53 genes and microsatellite instability in colonic adenocarcinomas arising in this model. Mutations of the Apc and p53 genes in the regions corresponding to mutation hot spots in human colorectal cancer were detd. by sequencing in 11 colonic adenocarcinomas. Microsatellite instability was detd. in matched normal and neoplastic DNA at five loci. All 11 adenocarcinomas harbored Apc mutations. Of these 11 tumors, 5 harbored truncating mutations. A total of 67 Apc mutations were found in these 11 tumors; 59 were missense mutations, whereas 8 were frameshift or nonsense

mutations. Six of the 11 adenocarcinomas harbored p53 mutations. A total of seven p53 mutations were found in these 11 tumors; all mutations were transitions, 4 of which were C:G.fwdarw.T:A transitions occurring in codon 229 at cytosine-guanine dinucleotides. Nine adenocarcinomas exhibited microsatellite instability in at least one of the five loci examd.; 1 tumor had microsatellite instability in two loci. Mol. genetics, as well as clin. features, of colon cancer in the .beta.2mnull .times. IL-2null mice are similar to those of human UC-assocd. colorectal cancer. As such, this model appears to be an excellent animal model to study UC-assocd. colorectal carcinogenesis.

REFERENCE COUNT: 63
REFERENCE(S):
(1) Andley, U; FASEB J 2001, V15, P221 CAPLUS
(2) Baker, S; Cell 1995, V82, P309 CAPLUS
(4) Berg, D; J Clin Investig 1996, V98, P1010 CAPLUS
(5) Beroud, C; Nucleic Acids Res 1996, V24, P121 CAPLUS
(6) Bienz, B; EMBO J 1984, V3, P2179 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 3 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001396706 EMBASE
TITLE: High prevalence of Mycoplasma pneumoniae in intestinal mucosal biopsies from patients with inflammatory bowel disease and controls.
AUTHOR: Chen W.; Li D.; Paulus B.; Wilson I.; Chadwick V.S.
CORPORATE SOURCE: W. Chen, Wakefield Gastroent. Res. Inst., Rintoul Street, Wellington 6039, New Zealand
SOURCE: Digestive Diseases and Sciences, (2001) 46/11 (2529-2535). Refs: 48
ISSN: 0163-2116 CODEN: DDSCDJ
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
005 General Pathology and Pathological Anatomy
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Intestinal microflora are believed to play an important role in the pathogenesis of inflammatory bowel disease (IBD). Mycoplasma have been suggested previously as organisms of ubiquitous distribution with the potential to cause inflammatory diseases, including IBD in susceptible individuals. The aim of this study was to determine the frequency of the presence of M. pneumoniae DNA in intestinal biopsies from patients with IBD and non-IBD controls using a microplate polymerase chain reaction-hybridization assay (PCR-ELISA). A total of 260 endoscopic biopsies (49 from 19 patients with Crohn's disease, 76 from 27 patients with **ulcerative colitis**, and 135 from 43 non-IBD controls) were used in this study. Overall, M. pneumoniae-specific DNA was detected in 100 endoscopic biopsy samples (38.5%). Among them, the detection rate of M. pneumoniae DNA was significantly higher in biopsies from patients with CD (59.2%) than in those from patients with UC (26.3%) or non-IBD controls (37.7%) ((X)(2) = 13.65, P .ltoreq. 0.001). The high prevalence of M. pneumoniae in both IBD patients and controls suggest this organism is ubiquitous and may persist in the intestinal mucosa. Epidemiological studies in IBD suggest acquisition of some agents early in life probably during epidemics in temperate latitudes. M. pneumoniae could be one of the ubiquitous agents implicated in the pathogenesis of IBD.

L246 ANSWER 4 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2001108811 EMBASE

TITLE: Dietary fiber fraction of germinated barley foodstuff attenuated mucosal damage and diarrhea, and accelerated the repair of the colonic mucosa in an experimental **colitis**.

AUTHOR: Kanauchi O.; Iwanaga T.; Andoh A.; Araki Y.; Nakamura T.; Mitsuyama K.; Suzuki A.; Hibi T.; Bamba T.

CORPORATE SOURCE: Dr. O. Kanauchi, Applied Bioresearch Center, Corporate Res./Development Division, Kirin Brewery Co. Ltd, Miyaharacho 3, Takasaki, Gunma 370-12, Japan. kanauchio@kirin.co.jp

SOURCE: Journal of Gastroenterology and Hepatology, (2001) 16/2 (160-168).
Refs: 38
ISSN: 0815-9319 CODEN: JGHEEO

COUNTRY: Australia
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 029 Clinical Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background and Aims: Germinated barley foodstuff (GBF) contains protein and insoluble dietary fiber. We have previously shown in **ulcerative colitis** patients and a colitis model that GBF feeding attenuates mucosal damage by increasing luminal **butyrate** levels. However, the detailed mechanism remains unclear because of its heterogeneous nature. The present study was carried out to: (i) evaluate the active ingredient in GBF; and (ii) examine its effect on the repair process in colonic inflammation by using a dextran sulfate sodium (DSS) colitis model. Methods: Colitis was induced by feeding a diet containing 0.5-3.5% DSS to male Sprague-Dawley rats. (i) Active ingredient: GBF was fractionated enzymatically into fiber- and protein-rich fractions. Each fraction was administered to DSS-colitis rats. Clinical signs, cecal short chain fatty acid concentrations and serum .alpha.(1)-acid glycoprotein (AAG) levels were determined. (ii) Effect on mucosal repair: GBF with or without salazosulfapyridine (SASP), or SASP alone was administered to rats after the onset of colitis. Seven days after initial treatment, the number of epithelial cells in HE sections was evaluated morphologically in a blind fashion and serum AAG was determined. Results: (i) Germinate barley foodstuff and GBF-fiber significantly attenuated the clinical signs of colitis and decreased serum AAG levels, with a significant increase in cecal **butyrate** production, while GBF-protein did not. (ii) Treatment with GBF alone and GBF plus SASP significantly accelerated colonic epithelial repair and improved clinical signs. Conclusions: These findings suggest that the fiber fraction of GBF may effectively enhance luminal **butyrate** production, and thereby accelerate colonic epithelial repair in colitis. .COPYRGT. 2001 Blackwell Science Asia Pty Ltd.

L246 ANSWER 5 OF 120 MEDLINE

ACCESSION NUMBER: 2001125183 MEDLINE
DOCUMENT NUMBER: 20567672 PubMed ID: 11115835

TITLE: Bacteria as the cause of ulcerative colitis.

AUTHOR: Campieri M; Gionchetti P

CORPORATE SOURCE: Centre for Inflammatory Bowel Disease, Department of Internal Medicine,University of Bologna Ospedale S. Orsola-Malpighi Via Massarenti, 9 40138 Bologna Italy.. campieri@med.unibo.it

SOURCE: GUT, (2001 Jan) 48 (1) 132-5. Ref: 70
Journal code: FVT. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom

Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010222

L246 ANSWER 6 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:578411 CAPLUS
DOCUMENT NUMBER: 135:204840
TITLE: The new experimental **ulcerative colitis** model in rats induced by subserosal injection of acetic acid
AUTHOR(S): Kojima, Ryotaro; Hamamoto, Shoichi; Moriwaki, Masahiko; Iwadate, Katsuharu; Ohwaki, Tatsuya
CORPORATE SOURCE: Res. Lab., Nisshin Kyorin Pharm. Co., Ltd., 5-3-1, Tsurugaoka, Oi-machi, Iruma-gun, Saitama, 356-8511, Japan
SOURCE: Nippon Yakurigaku Zasshi (2001), 118(2), 123-130
CODEN: NYKZAU; ISSN: 0015-5691
PUBLISHER: Nippon Yakuri Gakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB We have developed a new exptl. ulcerative colitis model in rats. Topical pathol. change of a round or a ellips shape was induced by subserosal injection of acetic acid (20%, 0.02 mL) into the middle colon of rats. The size of the induced ulcer could directly be measured using a caliper gauge, and the result was expressed as the ulcer area (mm2). We detd. the concn. of leukotriene B4 (LTB4), which is one of important clin. factors, in the ulcer region and found that the quantity of LTB4 was well correlated with the size of the ulcer area. Histopathol. studies of the ulcer region demonstrated that there were some morphol. similarities to the human form of ulcerative colitis, characterized by edema, necrosis, inflammatory cell infiltration, crypt abscess, and granulation tissue formation. Effects of 5-aminosalicylic acid and sodium prednisolone phosphate were investigated by intrarectal administration in this colitis model. The predominant improvement of colitis was obtained from both treatments in the ranges of the clin. doses of each drug. In conclusion, we suggest that this colitis model provides a new way for quant. evaluation of the efficacy of new therapeutic agents for ulcerative colitis.

L246 ANSWER 7 OF 120 MEDLINE
ACCESSION NUMBER: 2001175442 MEDLINE
DOCUMENT NUMBER: 21169673 PubMed ID: 11270609
TITLE: Nitric oxide production and iNOS mRNA expression in mice induced by repeated stimulation with live Fusobacterium nucleatum.
AUTHOR: Kato C; Mikami M; Saito K
CORPORATE SOURCE: Department of Oral Microbiology, School of Dentistry, Nippon Dental University at Niigata, Niigata, Japan.. ckato@ngt.ndu.ac.jp
SOURCE: MICROBIOLOGY AND IMMUNOLOGY, (2001) 45 (1) 69-78.
Journal code: MX7; 7703966. ISSN: 0385-5600.
PUB. COUNTRY: Japan
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200110
ENTRY DATE: Entered STN: 20011022
Last Updated on STN: 20011022
Entered Medline: 20011018

AB There have been few studies on the detection of direct nitric oxide (NO) production and interferon-gamma (IFN-gamma) in vivo without using animal cell culture. We questioned whether NO and IFN-gamma could be produced at the site of infection. The peritoneal cavity of mice was used as the local infection model. NO and IFN-gamma in abdominal washings from these mice were measured directly at various times after injection of Fusobacterium nucleatum, a gram-negative rod periodontal pathogen. The mice were divided into three groups: those treated with live bacteria (LB), those treated with heat-killed bacteria (HKB) and those untreated: normal (N). These mice were compared on the basis of cell filtration, NO and IFN-gamma production by injection of live bacteria (LFn) or heat-killed bacteria (HKFn). In the LB group, the total cell number increased corresponding to an increase in neutrophils after injection of both LFn and HKFn. A low level of NO was constantly produced in abdominal washings, but a significant amount of NO was synthesized in the LB group only 12 hr to 24 hr after injection of LFn. At the same time iNOS enzyme activity and iNOS mRNA expression were detected. IFN-gamma, which may contribute to enhance NO production, was also secreted at a high level from peritoneal exudate cells (PEC) at 12 hr and 24 hr in the LB group by stimulation of LFn. At 12 hr and 24 hr, iNOS positive cells in the LB group by infection of LFn were identified and shown to contain mostly macrophages. These findings indicate that live bacteria play important roles in NO production by macrophages. It is suggested that NO may contribute to the inflammatory response during F. nucleatum infection in periodontitis.

L246 ANSWER 8 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:241886 CAPLUS
DOCUMENT NUMBER: 134:220793
TITLE: An electron microscopic analysis of the histopathological similarity between a mouse colitis model induced by Yersinia enterocolitica heat-shock protein 60 and human **ulcerative colitis**
AUTHOR(S): Kuwahara, Norihiro
CORPORATE SOURCE: Dep. Pathol., Omori Hosp., Toho Univ. Sch. Med., 6-11-1 Omorinishi, Ota-ku, Tokyo, 143-8541, Japan
SOURCE: Toho Igakkai Zasshi (2001), 48(1), 34-40
CODEN: TOIZAG; ISSN: 0040-8670
PUBLISHER: Toho Daigaku Igakkai
DOCUMENT TYPE: Journal
LANGUAGE: Japanese

AB Recently, I developed a mouse colitis model using heat-shock protein (Hsp) 60, one of the ulcerative colitis (UC) pathogens. To det. the model's value, I conducted an ultrastructural comparison of the model with that of UC patients. I administered Hsp antigens i.p. to B10A/SgSn mice, and then obsd. the ultrastructure of murine intestinal tissues after various periods of administration. Colonic biopsy specimens were obtained from endoscopically-normal area of ten patients with clin. mild UC. In mice treated with Hsp antigen, I obsd. dilatation of intercellular spaces, pyknosis, increased cellular d., small and large vacuoles in cytoplasm, and absence of perivascular reticulum fibers in the subepithelial layer. These lesions gradually worsened with time and showed the various phases of degeneration. Apoptosis in Peyer's patches and lymphoid follicles sometimes appeared. In endoscopically-normal area of the UC patients, I obsd. ultrastructural findings similar to those in mice after limited Hsp

antigen administration. By modifying the duration of response to Hsp antigen, this model more closely approximated UC patients at various phases. These results suggest that this improved model will be useful in examg. UC treatment and prevention strategies.

L246 ANSWER 9 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:493663 CAPLUS
DOCUMENT NUMBER: 133:99560
TITLE: Bifidobacterium in the treatment of inflammatory disease
INVENTOR(S): Collins, John Kevin; O'Sullivan, Gerald Christopher; O'Mahony, Liam; Shanahan, Fergus
PATENT ASSIGNEE(S): Enterprise Ireland (Trading as Bioresearch Ireland), Ire.; National University of Ireland, Cork
SOURCE: PCT Int. Appl., 64 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 3
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2000042168	A2	20000720	WO 2000-IE8	20000117
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
EP 1141235	A2	20011010	EP 2000-900789	20000117
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
NO 2001003429	A	20010827	NO 2001-3429	20010710

PRIORITY APPLN. INFO.:
IE 1999-33 A 19990115
IE 1999-782 A 19990920
WO 2000-IE8 W 20000117

AB A strain of Bifidobacterium isolated from resected and washed human gastrointestinal tract is significantly immunomodulatory following oral consumption in humans. The strain is useful in the prophylaxis and/or treatment of undesirable inflammatory activity, esp. gastrointestinal inflammatory activity such as inflammatory bowel disease or irritable bowel syndrome. The inflammatory activity may also be due to cancer.

L246 ANSWER 10 OF 120 CABA COPYRIGHT 2002 CABI DUPLICATE 1
ACCESSION NUMBER: 2000:153857 CABA
DOCUMENT NUMBER: 20001421809
TITLE: The human gut bacteria Bacteroides thetaiotaomicron and **Fusobacterium varium** produce putrescine and spermidine in cecum of pectin-fed gnotobiotic rats
AUTHOR: Noack, J.; Dongowski, G.; Hartmann, L.; Blaut, M.
CORPORATE SOURCE: German Institute of Human Nutrition Potsdam-Rehbrucke, Department of Gastrointestinal Microbiology, 14558 Bergholz-Rehbrucke, Germany.
SOURCE: Journal of Nutrition, (2000) Vol. 130, No. 5, pp. 1225-1231.
ISSN: 0022-3166

DOCUMENT TYPE: Journal
LANGUAGE: English
AB Pectin is a soluble indigestible polysaccharide that stimulates cecal polyamine formation in rats. Bacteroides and fusobacteria, two numerically dominant bacterial population groups in the large intestine, were found to synthesize in vitro high amounts of spermidine and putrescine. The purpose of this study was to elucidate the effect of pectin on the polyamine production by defined bacterial species in vivo. Germfree male Wistar rats (n=18) were randomly assigned to one of three treatments: (i) monoassociation with Bacteroides thetaiotaomicron+fiber-free diet; (ii) diassociation with B. thetaiotaomicron+**Fusobacterium varium**+fiber-free diet or (iii) diassociation with B. thetaiotaomicron+**F. varium**+fiber-free diet+10% pectin. The cecal contents of monoassociated rats fed fiber-free diet contained large amounts (1.51 plus or minus 0.21 micro mol/dry total cecum content) of spermidine which was the major polyamine. The cecum of diassociated rats fed the fiber-free diet contained even higher concentrations of spermidine (2.53 plus or minus 0.21 micro mol/dry total cecum content) and also putrescine, which was now the dominant polyamine (putrescine 0.32 plus or minus 0.28 vs. 3.01 plus or minus 0.28 micro mol/dry total cecum content; monoassociation vs. diassociation). Pectin consumption by diassociated rats led to an additional increase in the cecal concentrations of all polyamines: putrescine, spermidine and spermine were 40, 37 and 100%, respectively, higher in the diassociated rats consuming the pectin diet than in those consuming the pectin-free diet. Since the microbial counts in the cecum did not differ in the diassociated treatment groups, the elevated concentrations of polyamines observed in the pectin group must have been due to stimulated bacterial polyamine synthesis. The decline of individual polyamines from cecum to feces detected at the end of the study in all treatment groups and the high microbial counts in the cecum and in feces suggest that bacterial polyamines are absorbed in cecum and colon. Pectin stimulates intestinal microbes to synthesize large amounts of polyamines which may be utilized by the host.

L246 ANSWER 11 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:324961 CAPLUS
DOCUMENT NUMBER: 132:329731
TITLE: Combined oral sodium **butyrate** and mesalazine treatment compared to oral mesalazine alone in **ulcerative colitis**: randomized, double-blind, placebo-controlled pilot study
AUTHOR(S): Vernia, P.; Monteleone, G.; Grandinetti, G.; Villotti, G.; Di Giulio, E.; Frieri, G.; Marcheggiano, A.; Pallone, F.; Caprilli, R.; Torsoli, A.
CORPORATE SOURCE: Cattedra di Gastroenterologia 1, Universita La Sapienza, Rome, Italy
SOURCE: Dig. Dis. Sci. (2000), 45(5), 976-981
CODEN: DDSCDJ; ISSN: 0163-2116
PUBLISHER: Kluwer Academic/Plenum Publishers
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Butyrate represents the main source of energy for colonic epithelial cells; however, its availability/utilization is impaired in ulcerative colitis (UC). In the present randomized, double-blind, placebo-controlled pilot study, the safety and efficacy of colonic targeted oral sodium butyrate tablets, coated with a pH-dependent sol. polymer, have been evaluated in ulcerative colitis. Thirty patients with mild to moderate colitis underwent a six-week course of oral sodium butyrate (4 g/day) plus oral mesalazine (2.4 g/day), (Group A) or of oral mesalazine plus placebo (Group B). Clin., endoscopic, and histol. data were collected at the

beginning and the end of the study. Twenty-five patients completed the study (12 in group A, 13 in group B). No untoward side effects were reported. In group A, seven patients underwent remission and four improved; in Group B the nos. were 5 and 5, resp. After treatment, all clin. parameters had significantly improved in both treatment arms compared to pretreatment findings. The UC disease activity index (UCDAI) score decreased from 7.27.+-.2.02 to 2.58.+-.2.19 ($P < 0.05$) in the combined treatment group and from 6.07.+-.1.60 to 3.46.+-.1.98 ($P < 0.05$) in group B. The endoscopic and histol. scores also significantly improved after treatment in both groups ($P < 0.05$). The difference between the two treatment arms was not significant, but a significantly better improvement vs. baseline values ($P < 0.05$) was obsd. in the combined treatment group vs. the mesalazine group, when considering both the clin. index (.DELTA.9.58.+-.4.19 vs. 5.92.+-.3.48) and the UCDAI score (.DELTA.4.67.+-.2.19 vs. 2.54.+-.2.18). A more favorable trend, although not significant, was obsd. for all individual parameters in group A. In conclusion, results of the present pilot study indicate that oral butyrate is safe and well tolerated. These data also suggest that oral butyrate may improve the efficacy of oral mesalazine in active ulcerative colitis and prompt the need of a large scale investigation to confirm the present findings.

REFERENCE COUNT: 31
REFERENCE(S):
(1) Ashford, M; J Drug Targ 1994, V2, P241 CAPLUS
(2) Boffa, L; J Biol Chem 1981, V256, P9612 CAPLUS
(4) Breuer, R; Gut 1997, V40, P485 CAPLUS
(6) Cummings, J; Gut 1981, V22, P763 CAPLUS
(7) D'Argenio, G; Gastoenterology 1994, V106, P399 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 12 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000177129 EMBASE
TITLE: **Butyrate** and glucose metabolism by colonocytes in experimental **colitis** in mice.
AUTHOR: Ahmad M.S.; Krishnan S.; Ramakrishna B.S.; Mathan M.; Pulimood A.B.; Murthy S.N.
CORPORATE SOURCE: Dr. B.S. Ramakrishna, Wellcome Trust Research Laboratory, Dept. of Gastrointestinal Sciences, Christian Medical College Hospital, Vellore 632004, India
SOURCE: Gut, (2000) 46/4 (493-499).
Refs: 40
ISSN: 0017-5749 CODEN: GUTTAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background/aims - Impaired colonocyte metabolism of **butyrate** has been implicated in the aetiopathogenesis of **ulcerative colitis**. Colonocyte **butyrate** metabolism was investigated in experimental colitis in mice. Methods - Colitis was induced in Swiss outbred white mice by oral administration of 4% dextran sulphate sodium (DSS). Colonocytes isolated from colitic and normal control mice were incubated with [14C]**butyrate** or glucose, and production of 14CO2, as well as of intermediate metabolites (acetoacetate, hydroxybutyrate and lactate), was measured. The effect of different te concentrations on oxidation was also examined. Results - e oxidation (.mu.mol/h per mg protein; mean (SEM)) was cantly reduced in DSS colitis, values on day 7 of DSS

administration being 0.177 (0.007) compared with 0.406 (0.035) for control animals (p<0.001). Glucose oxidation (.mu.mol/h per mg protein; mean (SEM)) on day 7 of DSS administration was significantly higher than in controls (0.06 (0.006) v 0.027 (0.004), p<0.001). Production of .beta.-hydroxybutyrate was decreased and production of lactate increased in DSS colitis compared with controls. Increasing **butyrate** concentration from 10 to 80 mM enhanced oxidation in DSS colitis (0.036 (0.002) to 0.285 (0.040), p<0.001), although it continued to remain lower than in controls. Surface and crypt epithelial cells showed similar ratios of **butyrate** to glucose oxidation. When 1 mM DSS was added to normal colonocytes in vitro, it did not alter **butyrate** oxidation. The initial histological lesion of DSS administration was very patchy and involved crypt cells. Abnormal **butyrate** oxidation became apparent only after six days of DSS administration, at which time histological abnormalities were more widespread. Conclusions - Colonocyte metabolism of **butyrate**, but not of glucose, is impaired in DSS colitis, and may be important in pathophysiology. Histological abnormalities preceded measurable defects in **butyrate** oxidation.

L246 ANSWER 13 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000209436 EMBASE
TITLE: Preventive efficacy of **butyrate** enemas and oral administration of Clostridium butyricum M588 in dextran sodium sulfate-induced **colitis** in rats.
AUTHOR: Okamoto T.; Sasaki M.; Tsujikawa T.; Fujiyama Y.; Bamba T.; Kusunoki M.
CORPORATE SOURCE: T. Okamoto, Second Dept. of Internal Medicine, Shiga University of Medical Science, Tsukinowa-cho, Seta, Otsu 520-2152, Japan
SOURCE: Journal of Gastroenterology, (2000) 35/5 (341-346).
Refs: 21
ISSN: 0944-1174 CODEN: JOGAET
COUNTRY: Japan
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
030 Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Butyrate** enemas have been reported to be effective in **ulcerative colitis**. However, long-term use is difficult because of the troublesome procedure and the unpleasant smell. We therefore investigated the effects of the oral administration of Clostridium butyricum M588 (CBM588), an enterobacterium producing **butyrate**, in dextran sodium sulfate (DSS)-induced colitis in rats. First, we confirmed the effects of pre-treatment with a **butyrate** enema on DSS colitis. We then studied the efficacy of oral administration of CBM588 which was started 1 week prior to the induction of DSS colitis. In the CBM588 group, the ulcer index and myeloperoxidase (MPO) activity in the distal colon were significantly lower than in the control group. Proliferating cell nuclear antigen (PCNA) immuno-positive cells were increased around the ulcer in the CBM588 group. In regard to the contents of the cecum and colon, the proportions of Lactobacillus and Eubacterium were increased in the cecum in the CBM588 group. Further, there were significant increases of n-**butyrate**, propionate, and acetate concentrations in the cecum in the CBM588 group. These results indicated that the oral administration of CBM588 alleviated DSS-induced colitis, and may be useful instead of **butyrate** enema.

L246 ANSWER 14 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000365992 EMBASE
TITLE: Enteric bacteria, lipopolysaccharides and related cytokines in inflammatory bowel disease: Biological and clinical significance.
AUTHOR: Caradonna L.; Amati L.; Magrone T.; Pellegrino N.M.; Jirillo E.; Caccavo D.
CORPORATE SOURCE: Dr. E. Jirillo, Immunologia, Policlinico, Piazza G. Cesare 4, 70124 Bari, Italy. jirillo@midim.uniba.it
SOURCE: Journal of Endotoxin Research, (2000) 6/3 (205-214).
Refs: 126
ISSN: 0968-0519 CODEN: JENREB
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; General Review
FILE SEGMENT: 004 Microbiology
026 Immunology, Serology and Transplantation
030 Pharmacology
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB **Ulcerative colitis** (UC) and Crohn's disease (CD) [inflammatory bowel disease (IBD)] are both characterized by an exaggerated immune response at the gut associated lymphoreticular tissue level. Such an abnormal and dysregulated immune response may be directed against luminal and/or enterio bacterial antigens, as also supported by murine models of inflammatory bowel disease (IBD) caused by organisms such as Citrobacter rodentium and Helicobacter hepaticus. Bacterial endotoxins or lipopolysaccharides (LPS) have been detected in the plasma of IBD patients and an abnormal microflora and/or an increased permeability of the intestinal mucosa have been invoked as cofactors responsible for endotoxemia. At the same time, the evidence that phagocytosis and killing exerted by polymorphonuclear cells and monocytes and the T-cell dependent antibacterial' activity are decreased in IBD patients may also explain the origin of LPS in these diseases. In IBD, pro-inflammatory cytokines and chemokines have been detected in elevated amounts in mucosal tissue and/or in peripheral blood, thus suggesting a monocyte/macrophage stimulation by enteric bacteria and/or their constituents (e.g. LPS). On these grounds, in experimental models and in human IBD, anti-cytokine monoclonal antibodies and interleukin receptor antagonists are under investigation for their capacity to neutralize the noxious effects of immune mediators. Finally, the administration of lactobacilli is beneficial in human IBD and, in murine colitis, this treatment leads to a normalization of intestinal flora, reducing the number of colonic mucosal adherent and translocated bacteria.

L246 ANSWER 15 OF 120 MEDLINE
ACCESSION NUMBER: 2001118209 MEDLINE
DOCUMENT NUMBER: 21069989 PubMed ID: 11154401
TITLE: Polarization of Porphyromonas gingivalis-specific helper T-cell subsets by prior immunization with Fusobacterium nucleatum.
AUTHOR: Choi J I; Borrello M A; Smith E S; Zauderer M
CORPORATE SOURCE: Cancer Center, Division of Immunology, School of Medicine and Dentistry, University of Rochester, New York, USA.
SOURCE: ORAL MICROBIOLOGY AND IMMUNOLOGY, (2000 Jun) 15 (3) 181-7.
Journal code: ORA. ISSN: 0902-0055.
PUB. COUNTRY: Denmark
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Dental Journals
ENTRY MONTH: 200102
ENTRY DATE: Entered STN: 20010322
Last Updated on STN: 20010322
Entered Medline: 20010215

AB Antigen-specific T-cell clones were obtained from mice immunized with Fusobacterium nucleatum ATCC 10953 and/or Porphyromonas gingivalis 381. 10 BALB/c mice per group were immunized with F. nucleatum followed by P. gingivalis, or with P. gingivalis alone by intraperitoneal injection of viable microorganisms. Spleen T cells were isolated and stimulated in vitro with viable P. gingivalis cells to establish P. gingivalis-specific T-cell clones. T-cell phenotypes and cytokine profiles were determined along with T-cell responsiveness to F. nucleatum or P. gingivalis. Serum immunoglobulin G antibody titers to F. nucleatum or P. gingivalis were also determined by enzyme-linked immunosorbent assay. All the T-cell clones derived from mice immunized with F. nucleatum followed by P. gingivalis demonstrated Th2 subsets, while those from mice immunized with P. gingivalis alone demonstrated Th1 subsets based on the flow cytometric analysis and cytokine profiles. All T-cell clones from both groups were cross-reactive to both P. gingivalis and F. nucleatum antigens. Phenotypes of T-cell clones were all positive for CD4. Mean post-immune serum IgG antibody levels to F. nucleatum or P. gingivalis were significantly higher than the pre-immune levels ($P < 0.05$, $P < 0.01$, respectively). There were no significant differences in the antibody titers between the two groups. It was concluded that P. gingivalis-specific T cells initially primed by cross-reactive F. nucleatum antigens were polarized to Th2 subset, while T cells stimulated with P. gingivalis alone maintained the profile of Th1 subset.

L246 ANSWER 16 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:257152 CAPLUS
DOCUMENT NUMBER: 133:265281
TITLE: Spontaneous chronic colitis in TCR.alpha.-mutant mice; an experimental model of human **ulcerative colitis**
AUTHOR(S): Bhan, Atul K.; Mizoguchi, Emiko; Smith, Rex Neal; Mizoguchi, Atsushi
CORPORATE SOURCE: Immunopathology Unit, Department of Pathology, Massachusetts General Hospital, Boston, MA, 02114, USA
SOURCE: Int. Rev. Immunol. (2000), 19(1), 123-138
CODEN: IRIMEH; ISSN: 0883-0185
PUBLISHER: Harwood Academic Publishers
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review with 66 refs. Mice with targeted disruption of the T cell receptor .alpha. gene (TCR.alpha.-/-) spontaneously develop chronic colitis. Colonic inflammation begins at 6-8 wk of age and chronic colitis is established in about 60% of mice by 16-20 wk of age. The disease is also assocd. with autoantibodies (anti-tropomyosin antibodies, anti-neutrophil cytoplasmic antibodies) and an oligoclonal immune response to luminal bacterial antigens. Although T cells, but not B cells or autoantibodies, are essential for the development of colitis, B cells and/or autoantibodies may have a regulatory role in the pathogenesis of this colitis because the colitis is more severe in B cell deficient TCR.alpha.-/- mice. Cytokines, specifically IL-4 and IL-1, also play an important role in the development of colitis in TCR.alpha.-/- mice. Enteric bacteria located in the large intestine are an important factor in the pathogenesis of this colitis because germ-free TCR.alpha.-/- mice do not develop colitis and appendectomy at an early age delays the onset of this colitis. The colitis in TCR.alpha.-/- mice resembles human

ulcerative colitis and provides a useful model to study the pathogenesis of human inflammatory bowel disease.

REFERENCE COUNT: 66
REFERENCE(S):
(1) Aranda, R; J Immunol 1997, V158, P3464 CAPLUS
(2) Baum, H; Immunol Today 1996, V17, P64 CAPLUS
(3) Berg, D; J Clin Invest 1996, V98, P1010 CAPLUS
(4) Berlin, C; Cell 1993, V74, P185 CAPLUS
(6) Bruno, L; Eur J Immunol 1995, V25, P1877 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 17 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:74694 CAPLUS
DOCUMENT NUMBER: 133:41508
TITLE: In vivo measurement of colonic **butyrate** metabolism in patients with quiescent **ulcerative colitis**
AUTHOR(S): Simpson, E. J.; Chapman, M. A. S.; Dawson, J.; Berry, D.; Macdonald, I. A.; Cole, A.
CORPORATE SOURCE: School of Biomedical Science, Nottingham University, Sutton Coldfield, B75 7RR, UK
SOURCE: Gut (2000), 46(1), 73-77
CODEN: GUTTAK; ISSN: 0017-5749
PUBLISHER: BMJ Publishing Group
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Background-Butyrate, a short chain fatty acid produced by bacterial fermn., is a major fuel source for the colonocyte. In vitro work has shown that ulcerative colitis may be characterized by a metabolic defect in colonocyte butyrate oxidn. Aims-To investigate the rate of metab. of rectally administered butyrate in patients with quiescent colitis. Methods-[1-13C]-butyrate enemas were administered to 11 patients with long standing quiescent ulcerative colitis and to 10 control patients. The rate of prodn. of 13CO2 in exhaled breath over four hours was measured by isotope ratio mass spectrometry combined with indirect calorimetry in order to measure CO2 prodn. This allowed calcn. of the patients' resting energy expenditure and RQ. Results-Over a four hour period, 325 (SEM 21) .mu.mol 13CO2 was recovered in breath samples from the colitis group compared with 322 (17) .mu.mol from the control group (NS). The RQ of the colitic group was significantly lower than that of the control group. Conclusion-There was no difference in the rate of metab. of butyrate between the two groups. It is unlikely that there is a primary metabolic defect of butyrate metab. in patients with quiescent ulcerative colitis.

REFERENCE COUNT: 25
REFERENCE(S):
(5) Clausen, M; Gut 1995, V37, P684 CAPLUS
(6) Corfield, A; Biochem Soc Trans 1992, V20, P95S CAPLUS
(7) Cummings, J; Gut 1981, V22, P763 CAPLUS
(8) Den Hond, E; Gastroenterology 1998, V115, P584 CAPLUS
(15) Mascolo, N; Gastroenterology 1991, V101, P331 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 18 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2001:671554 CAPLUS
DOCUMENT NUMBER: 135:366252
TITLE: Therapy of acute and severe **ulcerative colitis**
AUTHOR(S): Lengyel, Gabriella; Feher, Janos
CORPORATE SOURCE: 2nd Department of Medicine, Semmelweis University,

Budapest, H - 1088, Hung.
SOURCE: Prog. Hepato-Pharmacol. (2000), 5(Chronic Inflammatory Bowel Disease), 69-74
CODEN: PHPHFB; ISSN: 1335-4957
PUBLISHER: Liver and Drug Foundation
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with refs. Ulcerative colitis is a chronic disease of unknown etiol. characterized by inflammation of mucosa and submucosa of the large intestine, management of the patients with ulcerative colitis requires a comprehensive review of the patient's medical, nutritional and psychol. needs. The basic questions in the choice of the treatment for ulcerative colitis are: (a) the precise diagnosis, (b) the localization and complications of the disease, as well as (c) the activity of the inflammatory process. The pharmacotherapy in ulcerative colitis depends mainly on items sub b and c. Sulphasalazine, 5-aminosalicylic acid preparates and glucocortico-steroids are the most used antiinflammatory drugs. In the last years the topically acting steroids, immunosuppressants, immunomodulatory agents (cytokines, anticytokines) have been introduced in the acute forms of ulcerative colitis. Successful treatment depends on prompt recognition, early surgical consultation, and intensive resuscitative, antibacterial, and anti-inflammatory therapy. Important measures include i.v. fluids, plasma, blood, nasogastric suction, antibiotics, and i.v. corticosteroids. An early decision between intensive medical treatment and immediate surgery may be necessary, particularly if there is an evidence of perforation or peritonitis, or an uncontrollable hemorrhage.
REFERENCE COUNT: 7
REFERENCE(S):
(1) Actis, G; IBD at the end of its first century 2000, P199
(2) Compston, J; Aliment Pharmacol Ther 1995, V9, P237 MEDLINE
(3) Hanaeur, S; IBD at the end of its first century 2000, P178
(4) Hanaeur, S; N Engl J Med 1996, V334, P841
(7) Sheth, S; Lancet 1998, V351, P509 MEDLINE
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 19 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 2000:584277 CAPLUS
DOCUMENT NUMBER: 133:264999
TITLE: Morphological changes of lymphatic vessels in dextran sulfate sodium-induced **ulcerative colitis** of guinea pigs
AUTHOR(S): Ichikawa, Sanae
CORPORATE SOURCE: Department of Anatomy, Tokyo Medical University, Tokyo, 160-8402, Japan
SOURCE: Biomed. Res. (2000), 21(2), 57-65
CODEN: BRESD5; ISSN: 0388-6107
PUBLISHER: Biomedical Research Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Morphol. changes of lymphatic vessels in the cecum and ascending colon in dextran sulfate-induced ulcerative colitis of the guinea pig were examd. by light and electron microscopy. The lymphatic endothelium in the inflammatory cecum was composed of a mixt. of electron-lucent cells with large nos. of small vacuoles, lysosomes and developed ribosomes and electron-dense cells with ribosomes and rough endoplasmic reticulum. The lymphatic vessels displayed partially swollen endothelial cells with electron-lucent cytoplasm and indistinct mitochondrial cristae. The

lymphatic endothelium extended conspicuously irregular projections on the luminal surface, contg. pinocytotic vesicles. Lymphatic vessels full of infiltrating cells within the lumen appeared in the lamina propria of the initial part of the ascending colon and extended distally, but were not recognized in the cecum. These findings indicate that the morphol. changes of lymphatic vessels and the full of infiltrating cells within the lymphatic lumen may play an important role in eliminating and/or maintaining acute inflammatory processes by active permeation of immunoreactive substance into the lumen in this exptl. colitis model.

REFERENCE COUNT: 12
REFERENCE(S):
(2) Hoshi, O; J Gastroenterol 1996, V31, P189 CAPLUS
(3) Ichikawa, S; Arch Histol Cytol 1996, V59, P87 MEDLINE
(4) Ichikawa, S; Biomed Res 1998, V19, P261 CAPLUS
(5) Ichikawa, S; Lymphology 1987, V20, P73 MEDLINE
(11) Rice, G; Am J Pathol 1991, V138, P385 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 20 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:626055 CAPLUS
DOCUMENT NUMBER: 131:262591
TITLE: Treatment of **ulcerative colitis** with tropomyosin isoforms and monoclonal antibodies to tropomyosin isoforms
INVENTOR(S): Das, Kiron M.
PATENT ASSIGNEE(S): University of Medicine & Dentistry of New Jersey, USA
SOURCE: PCT Int. Appl., 38 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9948508	A1	19990930	WO 1999-US6193	19990322
W: AL, AM, AT, AU, AZ, BB, BG, BR, BY, CA, CH, CN, CZ, DE, DK, EE, ES, FI, GB, GE, HU, IS, JP, KE, KG, KP, KR, KZ, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, TJ, TM, TR, TT, UA, UG, UZ, VN, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
AU 9931079	A1	19991018	AU 1999-31079	19990322
EP 1064006	A1	20010103	EP 1999-912781	19990322
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				

PRIORITY APPLN. INFO.: US 1998-46049 A 19980323
WO 1999-US6193 W 19990322

AB This invention pertains to a method for treating ulcerative colitis. Specifically, the method comprises orally or rectally administering to a human having ulcerative colitis a therapeutically effective amt. of an antibody which binds to a tropomyosin isoform assocd. with ulcerative colitis. In another embodiment, the invention pertains to a method for treating ulcerative colitis in a human which comprises the steps of (a) obtaining from a human a colon epithelial cell ext. contg. a tropomyosin isoform assocd. with ulcerative colitis; (b) purifying the tropomyosin isoform until the tropomyosin isoform is substantially homogeneous; (c) developing an antibody which binds to the tropomyosin isoform; and (d)

orally or rectally administering to a human having ulcerative colitis a therapeutically effective amt. of the antibody to bind to the tropomyosin isoform assocd. with ulcerative colitis. In yet another embodiment, the invention pertains to a method for treating ulcerative colitis in a human which comprises orally administering to the human a therapeutically effective amt. of a tropomyosin isoform assocd. with ulcerative colitis.

REFERENCE COUNT: 2
REFERENCE(S): (1) Biancone; Clin Exp Immunol 1998, V113, P198 CAPLUS
(2) University of Medicine & Dentistry; WO 96/35449 A1 1996 CAPLUS

L246 ANSWER 21 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:594985 CAPLUS
DOCUMENT NUMBER: 131:223471
TITLE: Diagnosis, prevention and treatment of **ulcerative colitis**, and clinical subtypes thereof, using microbial **ulcerative colitis** pANCA antigens
INVENTOR(S): Braun, Jonathan; Cohavy, Offer
PATENT ASSIGNEE(S): The Regents of the University of California, USA
SOURCE: PCT Int. Appl., 134 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 9945955	A1	19990916	WO 1999-US5492	19990312
W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
RW: GH, GM, KE, LS, MW, SD, SL, SZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
US 6033864	A	20000307	US 1998-41889	19980312
AU 9930026	A1	19990927	AU 1999-30026	19990312
EP 1069907	A1	20010124	EP 1999-911375	19990312
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, NL, SE, PT, IE, FI				

PRIORITY APPLN. INFO.:

US 1998-41889	A	19980312
US 1996-57846	P	19960412
US 1997-837058	A2	19970411
WO 1999-US5492	W	19990312

AB The present invention relates to microbial ulcerative colitis (UC) perinuclear antineutrophil cytoplasmic autoantibodies (pANCA) antigens. The invention provides methods of diagnosing ulcerative colitis and methods of inducing tolerance in a pANCA-pos. patient with UC using a histone H1-like antigen. The invention further provides methods of diagnosing UC and methods of inducing tolerance in a pANCA-pos. patient with UC using a porin antigen. Methods of diagnosing UC and methods of inducing tolerance in a pANCA-pos. patient with UC using a Bacteroides antigen are also provided.

REFERENCE COUNT: 4
REFERENCE(S): (1) Cohavy; FASEB Journal 1998, V12(4), PA593
(2) Eggena; FASEB Journal 1996, V10(6), PA1079
(3) Sartor; American Journal of Gastroenterology 1997,

V92(12), P5S
(4) The Regents Of The University Of California; WO 9738713 A1 1997 CAPLUS

L246 ANSWER 22 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:104418 CAPLUS
DOCUMENT NUMBER: 130:181477
TITLE: Method of treating **ulcerative colitis** with a monoclonal antibody
INVENTOR(S): Das, Kiron M.
PATENT ASSIGNEE(S): University of Medicine & Dentistry, USA
SOURCE: U.S., 9 pp., Cont.-in-part of U.S. Ser. No. 437,474, abandoned.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 2
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 5869048	A	19990209	US 1996-630541	19960410
WO 9635449	A1	19961114	WO 1996-US6589	19960509

W: CA, JP, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM

PRIORITY APPLN. INFO.: US 1995-437474 19950509
US 1996-630541 19960410

AB The present invention pertains to a method for treating ulcerative colitis in a human which comprises orally or rectally administering to the human a therapeutically effective amt. of an antibody which binds to a colonic antigen assocd. with ulcerative colitis. In another embodiment, the present invention pertains to a method for treating ulcerative colitis in a human which comprises the steps of (a) obtaining from a human a colon epithelial cell ext. contg. a colonic antigen assocd. with ulcerative colitis; (b) purifying the colonic antigen until the antigen is substantially homogeneous; (c) developing an antibody which binds to the colonic antigen; (d) orally or rectally administering to a human having ulcerative colitis a therapeutically effective amt. of the antibody to bind to the colonic antigen assocd. with ulcerative colitis. In yet another embodiment, the present invention pertains to a method for vaccinating a human against ulcerative colitis which comprises orally administering to the human a therapeutically effective amt. of a colonic antigen assocd. with ulcerative colitis. The colon epithelial antigen is p40 or gp185, and the monoclonal antibody is 7E12H12.

REFERENCE COUNT: 1
REFERENCE(S): (1) Nandiwada; Gastroenterol 1993, V104, PA754

L246 ANSWER 23 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 2000022542 EMBASE
TITLE: Germinated barley foodstuffs attenuate colonic mucosal damage and mucosal nuclear factor kappa B activity in a spontaneous **colitis** model.
AUTHOR: Kanauchi O.; Andoh A.; Iwanaga T.; Fujiyama Y.; Mitsuyama K.; Toyonaga A.; Bamba T.
CORPORATE SOURCE: Dr. O. Kanauchi, Applied Bioresearch Center, Corporate Res. and Devt. Division, Kirin Brewery Co. Ltd., Miyaharacho 3, Takasaki, Gunma 370-1295, Japan. kanauchio@kirin.co.jp
SOURCE: Journal of Gastroenterology and Hepatology, (1999) 14/12 (1173-1179).
Refs: 31
ISSN: 0815-9319 CODEN: JGHEEO

COUNTRY: Australia
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background: Germinated barley foodstuffs (GBF), which are derived from brewer's spent grain and are a highly safe food substance, increased **butyrate** production in the lower intestine and prevented mucosal damage and bloody diarrhoea in an acute experimental colitis model. As human histocompatibility leucocyte antigen (HLA)-B27 transgenic rats develop spontaneous and chronic intestinal inflammation resembling **ulcerative colitis**, we investigated the mechanisms underlying the preventive effects of GBF against a spontaneous and chronic colitis model. Specifically, the production of bacterial **butyrate** and the regulation of proinflammatory cytokine production were examined. Methods: A GBF diet and a cellulose (CE) diet were fed to HLA-B27 transgenic rats for 13 weeks. The presence of faecal occult blood, colonic mucosal protein, DNA and RNA content, colonic myeloperoxidase activity, nuclear factor kappa B (NF.kappa.B) DNA binding activity, the depth of the crypts and serum inflammatory parameters were then evaluated. **Butyrate** production in the caecal contents was also determined. Results: Feeding GBF significantly increased bacterial **butyrate** production and simultaneously attenuated the presence of faecal occult blood and colonic mucosal hyperplasia. Colonic mucosal NF.kappa.BDNA binding activity and the production of interleukin-8 were also suppressed by the **butyrate** produced from GBF. Conclusions: Germinated barley foodstuffs feeding promotes bacterial **butyrate** production and attenuated inflammation in both spontaneous and chronic colitis in HLA-B27 transgenic rats.

L246 ANSWER 24 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1999:329083 BIOSIS
DOCUMENT NUMBER: PREV199900329083
TITLE: Pathological evaluation of utility of mouse colitis induced by Yersinia enterocolitica heat-shock protein 60 for investigating **ulcerative colitis**: Comparing with existing colitis **models**.
AUTHOR(S): Ihara, T. (1); Sugamata, Masao (1); Yamamoto, T. (1); Kuwahata, N.; Hasegawa, C.; Miura, M.; Toda, T.
CORPORATE SOURCE: (1) Tochigi Institute of Clin Pathology, Tochigi Japan
SOURCE: Gastroenterology, (April, 1999) Vol. 116, No. 4 PART 2, pp. A741.
Meeting Info.: Digestive Disease Week and the 100th Annual Meeting of the American Gastroenterological Association Orlando, Florida, USA May 16-19, 1999 American Gastroenterological Association
. ISSN: 0016-5085.
DOCUMENT TYPE: Conference
LANGUAGE: English

L246 ANSWER 25 OF 120 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 2001:128322 CABA
DOCUMENT NUMBER: 20013048196
TITLE: Characteristics of **Fusobacterium** ulcerans, a new and unusual species compared with **Fusobacterium varium** and **Fusobacterium** mortiferum
AUTHOR: Claros, M. C.; Papke, Y.; Kleinkauf, N.; Adler, D.; Citron, D. M.; Hunt-Gerardo, S.; Montag, Th.; Goldstein, E. J. C.; Rodloff, A. C.; Finegold, S. M.

[EDITOR]; Citron, D. M. [EDITOR]; Goldstein, E. J. C. [EDITOR]
SOURCE: Anaerobe, (1999) Vol. 5, No. 3/4, pp. 137-140. 8 ref.
Meeting Info.: Proceedings of the Anaerobic Society of the Americas Congress on Anaerobic Bacteria and Anaerobic Infections, Buenos Aires, Argentina, 24-26 April, 1998.
ISSN: 1075-9964
DOCUMENT TYPE: Journal; Conference Article
LANGUAGE: English

L246 ANSWER 26 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:67846 CAPLUS
DOCUMENT NUMBER: 130:280175
TITLE: Changes in plasma and colonic mucosa fatty acid profiles in rats with **ulcerative colitis** induced by trinitrobenzene sulfonic acid
AUTHOR(S): Nieto, N.; Giron, M. D.; Suarez, M. D.; Gil, A.
CORPORATE SOURCE: Department of Biochemistry and Molecular Biology, School of Pharmacy, University of Granada, Granada, 18071, Spain
SOURCE: Dig. Dis. Sci. (1998), 43(12), 2688-2695
CODEN: DDSCDJ; ISSN: 0163-2116
PUBLISHER: Plenum Publishing Corp.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Polyunsatd. fatty acids have a key role in the pathogenesis of inflammatory bowel disease since some of the arachidonic acid-derived eicosanoids have been found to be increased in inflamed intestinal mucosa in the acute phase of human disease. The aim of this study was to prospectively assess plasma and colon mucosa fatty acid patterns in rats with exptl. ulcerative colitis. Twenty rats were treated with trinitrobenzene sulfonic acid and 20 with NaCl; two groups were killed after one week and two after two weeks to evaluate colon damage. Plasma was obtained by aortic puncture and colonic mucosa was scraped off and the fatty acid pattern was detd. by gas-liq. chromatog. Total, satd., and monounsatd. plasma fatty acids were significantly higher in both periods of ulcerative colitis as compared to controls. Plasma n-6 fatty acids were increased after treatment, but no significant changes were obsd. concerning to n-3 fatty acids. With regard to colon mucosa, satd. and monounsatd. fatty acids did not change because of the disease; however, n-6 fatty acids decreased in the first week and increased in the second week and n-3 fatty acids were increased. Changes on the fatty acid distribution in plasma did not parallel to those of colonic mucosa except for 22:6(n-3). We have also found that exptl. ulcerative colitis induced by trinitrobenzene sulfonic acid reproduces many of the features related to changes in plasma and colon mucosa fatty acids obsd. in the human disease.

REFERENCE COUNT: 36
REFERENCE(S):
(1) Adam, O; J Lipid Res 1986, V27, P421 CAPLUS
(2) Brenner, R; Prog Lipid Res 1981, V20, P41 CAPLUS
(3) Budowski, P; Proc Nutr Soc 1985, V44, P221 CAPLUS
(6) Donowitz, M; Gastroenterology 1985, V88, P580 CAPLUS
(11) Falardeau, P; Biochim Biophys Acta 1976, V441, P193 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 27 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1999:44169 CAPLUS
DOCUMENT NUMBER: 130:208738
TITLE: Involvement of interleukin-1 in the development of **ulcerative colitis** induced by dextran sulfate sodium in mice
AUTHOR(S): Arai, Yoshinori; Takanashi, Hitoshi; Kitagawa, Hiroshi; Okayasu, Isao
CORPORATE SOURCE: Laboratory for Pharmacology, Preclinical Development Laboratories, Research and Development Division, Nippon Hoechst Marion Roussel Ltd., Kawagoe, 350-11, Japan
SOURCE: Cytokine (1998), 10(11), 890-896
CODEN: CYTIE9; ISSN: 1043-4666
PUBLISHER: Academic Press
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Dextran sulfate sodium (DSS)-induced colitis in mice has been recognized as a model for human ulcerative colitis. Using this model, the effects of anti-murine interleukin 1.beta. (IL-I.beta.) antibodies (anti-muIL-1.beta.) and recombinant murine IL-1 receptor type I (rmuIL-1R) on the development of colitis were examd. to det. whether IL-1 plays a role in colitis. Furthermore, RT-PCR amplification was used to examine for the presence of mRNAs for IL-1.alpha. and IL-1.beta. in the large intestine. In mice with colitis induced by DSS, administration of anti-muIL-1.beta. (5 mg/kg, once/wk, i.p.) significantly suppressed body wt. loss and shortening of the large intestine. Administration of rmuIL-1R (0.2 mg/kg or 1.0 mg/kg, once/day, i.v.) significantly suppressed shortening of the large intestine. Expression of mRNAs for IL-1.alpha. and IL-1.beta. was obsd. in the large intestine of mice which received distd. water contg. 3% DSS for 5 days. The expression tended to increase in mice which received DSS for 11 days. In contrast, mRNA expression was not obsd. in mice which received distd. water without DSS. These results clearly demonstrate that IL-1 is involved in the development of DSS-induced colitis in mice and suggest that downregulation of IL-1 might be useful for the treatment of patients with ulcerative colitis. (c) 1998 Academic Press.
REFERENCE COUNT: 41
REFERENCE(S):
(2) Ciancio, M; Ann NY Acad Sci 1992, V664, P210 CAPLUS
(3) Cominelli, F; Gastroenterology 1989, V97, P1400 CAPLUS
(4) Cominelli, F; Gastroenterology 1992, V103, P65 CAPLUS
(5) Cominelli, F; J Clin Invest 1990, V86, P972 CAPLUS
(6) Dieleman, L; Gastroenterology 1994, V107, P1643 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 28 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:611795 CAPLUS
DOCUMENT NUMBER: 130:23623
TITLE: In vivo **butyrate** metabolism and colonic permeability in extensive **ulcerative colitis**
AUTHOR(S): Den Hond, Elly; Hiele, Martin; Evenepoel, Pieter; Peeters, Marc; Ghoos, Yvo; Rutgeerts, Paul
CORPORATE SOURCE: Department of Gastroenterology, University Hospital Leuven, Louvain, Belg.
SOURCE: Gastroenterology (1998), 115(3), 584-590

CODEN: GASTAB; ISSN: 0016-5085
PUBLISHER: W. B. Saunders Co.
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Impaired short-chain fatty acid metab. by the colonocyte has been suggested as a pathogenic factor in ulcerative colitis (UC). The aim of this study was to measure in vivo butyrate metab. in UC and to correlate butyrate oxidn. with colonic permeability. Butyrate oxidn. was measured by a 14CO2-breath test after rectal instillation of 14C-butyrate. 51Cr-EDTA was added to the enema, and the urinary % dose excretion of 51Cr-EDTA after 6 h was a measure for permeability. Patients with active extensive UC showed a significantly lower butyrate oxidn. and increased colonic permeability in comparison to healthy controls. Butyrate oxidn. correlated significantly neg. with clin. activity. Oxidn. of butyrate was not decreased in most patients with inactive extensive UC. In 3 patients with inactive disease and decreased oxidn., a relapse occurred within a few weeks after the test, whereas all patients with normal oxidn. maintained their remission for at least 3 mo. A significantly neg. correlation existed between butyrate oxidn. and colonic permeability. Patients with active extensive UC have a decreased colonic butyrate oxidn. However, the fact that remission is assocd. with normal oxidn. suggests that UC mucosa is not intrinsically altered in butyrate oxidn., making this unlikely to be a primary defect in UC.
REFERENCE COUNT: 30
REFERENCE(S): (2) Breuer, R; Gut 1997, V40, P485 CAPLUS
(6) Clausen, M; Gut 1995, V37, P684 CAPLUS
(7) Cummings, J; Br J Nutr 1996, V75, P733 CAPLUS
(8) Cummings, J; Lancet 1983, V1, P1206 CAPLUS
(10) Evans, M; J Am Diet Assoc 1992, V92, P1239 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L246 ANSWER 29 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1998:780043 CAPLUS
DOCUMENT NUMBER: 130:235782
TITLE: Chronic **ulcerative colitis** model in rats
AUTHOR(S): Zheng, Li; Gao, Zhenqiang; Wang, Shuxian
CORPORATE SOURCE: Dept of Pharmacology, National Institutes of Pharmaceutical Research and Development, Beijing, 102206, Peop. Rep. China
SOURCE: Zhongguo Yaolixue Tongbao (1998), 14(4), 370-372
CODEN: ZYTOE8; ISSN: 1001-1978
PUBLISHER: Anhui Yike Daxue Linchuan Yaoli Yanjiuso
DOCUMENT TYPE: Journal
LANGUAGE: Chinese
AB Chronic ulcerative colitis model in rat was examd. by using trinitrobenzene sulfonic acid (TNBS) at various concn. The chronic inflammation of the rat colon was induced by a single colonic instillation of TNBS (25-150 mg kg-1) dissolved in 0.5 mL of 30% ethanol. Three wk after exposure to TNBS, the severity of colonic damage were assessed by gross appearance using a grading scale (0- 5), colon wts. and by histol. The no. of neutrophils present in inflamed colonic tissue was measured by the myeloperoxidase (MPO) assay. The relationship between MPO activity and time-course was studied. At a dose of 100-150 mg kg-1, TNBS/ethanol induced ulcer and thickened markedly the bowel wall until 7 wk, and significantly increased MPO activity. The inflammatory responses revealed in mucosal and submucosal infiltration by neutrophils, macrophages, lymphocytes and fibroblasts, granulomas formation and cryptoabscess were obsd. At TNBS dose of 50 mg kg-1 there was a lower level of colonic damage, the lowest dose of TNBS (25 mg kg-1) did not show significantly

different from control animals (P > 0.05). A TNBS dose of 100 mg kg-1 was chosen for an appropriate exptl. dose.

L246 ANSWER 30 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:677956 CAPLUS
DOCUMENT NUMBER: 127:329782
TITLE: Protease activity in a hapten-induced model of **ulcerative colitis** in rats
AUTHOR(S): Hawkins, James V.; Emmel, Eva L.; Feuer, Jennifer J.; Nedelman, Mark A.; Harvey, Catherine J.; Klein, Hilton J.; Rozmiarek, Harry; Kennedy, Ann R.; Lichtenstein, Gary R.; Billings, Paul C.
CORPORATE SOURCE: Laboratory Animal Medicine, Departments of Radiation Oncology and Pathology, Gastroenterology Division, Department of Medicine, School of Medicine, Department of Pathology, School of Dental Medicine, University of Pennsylvania, Philadelphia, PA, 19104, USA
SOURCE: Dig. Dis. Sci. (1997), 42(9), 1969-1980
CODEN: DDSCDJ; ISSN: 0163-2116
PUBLISHER: Plenum
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Inflammatory bowel disease (IBD) is a painful and debilitating condition affecting the mucosal lining of the colon and other areas of the gastrointestinal tract. IBD generally falls into two major categories: ulcerative colitis (UC) and Crohn's disease. The authors have utilized dinitrobenzenesulfonic acid (DNBS) to induce exptl. UC in rats. Histopathol. anal. indicates that DNBS induces a condition in animals similar to human UC. Biochem. results revealed 6- to 10-fold elevated levels of serine protease activity in colon tissue from animals with UC as compared with matched controls. The authors also obsd. elevated levels of protease activity in tissue samples obtained from human patients with UC. Hence, the authors' results demonstrate that protease activity is increased in rodent and human UC. These proteases may play a significant role in destruction of colonic tissue in IBD. Protease inhibitors that target serine proteases may be useful pharmacol. agents to limit tissue destruction in IBD.

L246 ANSWER 31 OF 120 MEDLINE
ACCESSION NUMBER: 97264399 MEDLINE
DOCUMENT NUMBER: 97264399 PubMed ID: 9110232
TITLE: Effect of **Fusobacterium** necrophorum leukotoxoid vaccine on susceptibility to experimentally induced liver abscesses in cattle.
AUTHOR: Saginala S; Nagaraja T G; Lechtenberg K F; Chengappa M M; Kemp K E; Hine P M
CORPORATE SOURCE: Department of Animal Sciences, Kansas State University, Manhattan 66506, USA.
SOURCE: JOURNAL OF ANIMAL SCIENCE, (1997 Apr) 75 (4) 1160-6.
Journal code: HC7; 8003002. ISSN: 0021-8812.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199706
ENTRY DATE: Entered STN: 19970716
Last Updated on STN: 19970716
Entered Medline: 19970627

AB The efficacy and the optimum dose of Fusobacterium necrophorum crude leukotoxoid vaccine required to immunize and protect steers against experimentally induced liver abscesses were evaluated. The vaccine consisted of cell-free culture supernatant of a high leukotoxin-producing strain of F. necrophorum, inactivated with formalin and homogenized with an adjuvant. Twenty-five steers were assigned randomly to the following five treatment groups: control; three doses (1.0, 2.0, and 5.0 mL) of the culture supernatant; and 2.25 mL of the concentrated supernatant (equivalent to 5 mL of the original supernatant). Vaccine was injected subcutaneously on d 0 and 21. Blood samples were collected weekly to monitor antileukotoxin antibody titers. Three weeks after the second vaccination (d 42), all steers were injected intraportally with F. necrophorum culture to induce liver abscesses. Three weeks later (d 63), steers were euthanatized and necropsied; livers were examined and protection assessed. Antileukotoxin antibody titers in the control steers generally did not differ from the baseline (wk 0) titers. The titers in the vaccinated groups increased, more so after the second injection, and the increase was generally dose-dependent. Necropsy examination revealed that all steers in the control group had abscesses in the liver. In the vaccinated groups, two of five steers in the 1.0-mL group and one each in the 2.0-, 5.0-, and 2.25-mL (concentrated) groups had liver abscesses. Antileukotoxin antibody titers were higher ($P < .05$) in steers that did not develop abscesses than in steers that developed abscesses. The difference suggested a protective effect of antileukotoxin antibodies against experimentally induced liver abscesses.

L246 ANSWER 32 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:412216 BIOSIS
DOCUMENT NUMBER: PREV199799704259
TITLE: Animal **models** for intra-abdominal infection.
AUTHOR(S): Sayek, Iskender
CORPORATE SOURCE: Hacettepe Univ., Dep. Surgery, Ankara Turkey
SOURCE: Hepato-Gastroenterology, (1997) Vol. 44, No. 16, pp. 923-926.
ISSN: 0172-6390.
DOCUMENT TYPE: Journal; Article
LANGUAGE: English
AB Animal **models** are widely used to study intra-abdominal infections. Experimental intra-abdominal infections. Experimental intra-abdominal infection is a bi-phasic process; initially a peritonitis or septic phase caused by E. coli and later a chronic-abscess forming stage, caused by B. fragilis similar to human beings. Various methods have been described. These methods includes challenge of the peritoneum with either endogenous bacteria or inoculation of pure bacteria or fecal material. Non-bacterial **models** have also been described. Each of these **models** have their own advantages and disadvantages. The aim and the end points should govern the selection of the right **model** to be used for experimental purposes. The ideal **model** should be reliable, standard, reproducible and resembling human disease. A single **model** with those specifications is yet to be described.

L246 ANSWER 33 OF 120 MEDLINE
ACCESSION NUMBER: 1998071224 MEDLINE
DOCUMENT NUMBER: 98071224 PubMed ID: 9407333
TITLE: Thiolmethyltransferase activity in the human colonic mucosa: implications for ulcerative **colitis**.
AUTHOR: Moore J W; Babidge W J; Millard S H; Roediger W E
CORPORATE SOURCE: University of Adelaide, Department of Surgery, Queen Elizabeth Hospital, Woodville, Australia.

SOURCE: JOURNAL OF GASTROENTEROLOGY AND HEPATOLOGY, (1997 Oct) 12 (9-10) 678-84.
Journal code: A6J; 8607909. ISSN: 0815-9319.
PUB. COUNTRY: Australia
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199801
ENTRY DATE: Entered STN: 19980206
Last Updated on STN: 19990129
Entered Medline: 19980127

AB Ulcerative colitis is associated with a selective reduction of n-butyrate oxidation by the colonic epithelial cells although the reason for this has been unclear. Colonic epithelial cell n-butyrate oxidation can be inhibited in vitro by incubation with sulphide but the role of mucosal detoxification of sulphide in the metabolic welfare of the colonic mucosa has not been examined. This study aimed to assess the role mucosal detoxification of sulphide by thiolmethyltransferase (TMT)-mediated methylation may play in protecting the healthy colonic mucosa from the adverse effects of luminal sulphide. Colonic epithelial cell suspensions from healthy human proximal (n = 9) and distal colon (n = 10) were incubated in the presence of 14C-labelled n-butyrate (5 mmol/L) alone, butyrate plus sodium hydrogen sulphide (NaHS) (1.5 mmol/L), or butyrate plus NaHS plus S-adenosyl-methionine 1,4 butane disulphonate (SAMe) (5 mmol/L). Study end points were metabolic performance (14CO2 production) and mucosal TMT activity. Incubation with NaHS induced a significant inhibition of 14CO2 production compared with control incubations ($P < 0.001$) which was similar for proximal and distal colonic cell suspensions. S-adenosyl-methionine 1,4 butane disulphonate reversed this effect completely in proximal but not in distal cell incubations, suggesting a greater susceptibility of the distal colon to the sulphide effect. Although median whole mucosal TMT values did not differ between proximal and distal colonic mucosa, a non-normal distribution of distal TMT values was observed. However, neither the degree of sulphide inhibition of control 14CO2 production nor the degree to which SAMe reversed this inhibition correlated with whole mucosal TMT activity. The study concluded that regional variation exists in TMT activity in the human colon but whilst methylation appears to protect colonic epithelial cells against sulphide-induced inhibition of n-butyrate oxidation, this cannot be directly correlated with mucosal TMT activity.

L246 ANSWER 34 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 97258942 EMBASE
DOCUMENT NUMBER: 1997258942
TITLE: Frequency of pathogenic and enteroadherent Escherichia coli in patients with inflammatory bowel disease and controls.
AUTHOR: Schultsz C.; Moussa M.; Van Ketel R.; Tytgat G.N.J.; Dankert J.
CORPORATE SOURCE: Dr. C. Schultsz, Department of Medical Microbiology, Academic Medical Centre, Meibergdreef 9, 1105 AZ Amsterdam, Netherlands. c.schultsz@amc.uva.nl
SOURCE: Journal of Clinical Pathology, (1997) 50/7 (573-579).
Refs: 38
ISSN: 0021-9746 CODEN: JCPAAK
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Aims - To determine whether inflammatory bowel disease (IBD) is associated with pathogenic or enteroadherent Escherichia cell. Methods - A least two stool specimens and one rectal biopsy were taken from 30 patients with IBD and from 20 controls. A large number of E coli-like colonies cultured from each stool sample and biopsy was tested, using DNA probes, for the presence of genes encoding shiga-like toxins, invasiveness, attachment-effacement and the ability to adhere to HEp-2 cells. Similarity among isolates from stool samples and rectal biopsies was determined by random amplified polymorphic DNA (RAPD) analysis. Results - Enterohaemorrhagic and enteroinvasive E coli were not found in samples from either patients or controls. No significant difference in the detection rate of enteroadherent E coli between patients and controls was found. Rectal biopsies from 11 of 28 patients with IBD and 4 of 18 controls contained E coli, which hybridised with probes for detection of genes encoding diffuse adherence to HEp-2 cells, or encoding P-pili (p = 0.2). Enteroadherent E coli isolated from two or three stool specimens from the same patient or control appeared to be identical by RAPD analysis, and are considered to be residents in the colon. Probe positive isolates obtained from stool specimens and corresponding rectal biopsies were always identical on RAPD analysis. Conclusions - E coli strains possessing adherence factors reside in the large intestine and adhere to the rectal mucosa, irrespective of the presence of colitis.

L246 ANSWER 35 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:186487 CAPLUS
DOCUMENT NUMBER: 126:210371
TITLE: Experimental **ulcerative colitis**
AUTHOR(S): Iwanaga, Toshihiko
CORPORATE SOURCE: Hokkaido Univ., Sapporo, 060, Japan
SOURCE: Igaku no Ayumi (1997), 180(6), 380-381
CODEN: IGAYAY; ISSN: 0039-2359
PUBLISHER: Ishiyaku
DOCUMENT TYPE: Journal; General Review
LANGUAGE: Japanese
AB A review with 5 refs., esp. on usefulness and mechanism of dextran sulfate (DDS)-induced ulcerative colitis as exptl. model, and species difference in sensitivity to DDS.

L246 ANSWER 36 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97169546 EMBASE
DOCUMENT NUMBER: 1997169546
TITLE: Recovery of Lactobacillus rhamnosus GG from human colonic biopsies.
AUTHOR: Alander M.; Korpela R.; Saxelin M.; Vilpponen-Salmela T.; Mattila-Sandholm T.; Von Wright A.
CORPORATE SOURCE: M. Alander, Research Scientist, VTT, Biotechnology and Food Research, PO Box 1501, FIN-0244 VTT, Finland
SOURCE: Letters in Applied Microbiology, (1997) 24/5 (361-364).
Refs: 13
ISSN: 0266-8254 CODEN: LAMIE7
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The colonization of Lactobacillus rhamnosus GG (ATCC 53103, henceforth L.GG) in five human colonoscopy patients was studied. The test subjects consumed whey drink fermented with the bacterium for 12 d before the colonoscopy. The presence of L.GG was subsequently checked both in the faecal samples and in the colonic biopsies obtained from various locations

in the large intestine. In all patients L.GG was the dominant faecal lactic acid bacterium as a result of the administration. In four patients L.GG could also be recovered from the biopsies, while with one patient (suffering from **ulcerative colitis** diagnosed during the colonoscopy) no L.GG was detected in the biopsy samples. The results suggest that L.GG is able to adhere in vivo to the colon. Study of the faecal samples alone is apparently not sufficient for elucidation of the gastrointestinal ecology of probiotic bacteria.

L246 ANSWER 37 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:488894 BIOSIS
DOCUMENT NUMBER: PREV199799788097
TITLE: Characterization of a novel bacteriophage in **Fusobacterium varium**.
AUTHOR(S): Andrews, D. M. A.; Gharbia, S. E.; Shah, H. N. (1)
CORPORATE SOURCE: (1) Dep. Microbiol., Eastman Dental Inst., 256 Gray's Inn Road, London WC1X 8LD UK
SOURCE: Clinical Infectious Diseases, (1997) Vol. 25, No. SUPPL. 2, pp. S287-S288.
ISSN: 1058-4838.
DOCUMENT TYPE: Article
LANGUAGE: English

L246 ANSWER 38 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:377161 CAPLUS
DOCUMENT NUMBER: 127:106375
TITLE: Pathogenesis and virulence of Rhodococcus equi
AUTHOR(S): Hondalus, Mary K.
CORPORATE SOURCE: Howard Hughes Medical Research Institute, Albert Einstein College of Medicine, Bronx, NY, 10461, USA
SOURCE: Vet. Microbiol. (1997), 56(3,4), 257-268
CODEN: VMICDQ; ISSN: 0378-1135
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review, with 77 refs. Inhalation of the soil-borne organism, Rhodococcus equi, can lead to a chronic and severe pyogranulomatous pneumonia in young horses and immunocompromised people. In addn., ulcerative colitis is a common sequela to infection in foals, and dissemination from the lung to other body sites is not uncommon in either the horse or man. Although the facultative intracellular bacterium is susceptible to neutrophil-mediated killing, it is able to resist innate macrophage defenses and establish residence within the intracellular environment of that phagocyte. Definitive virulence factors of R. equi have not yet been detd., but potential candidates include capsular polysaccharide, the exoenzyme cholesterol oxidase, cell wall mycolic acids, and the products encoded by a virulence-assocd. plasmid. The ability to replicate within the macrophage is assocd. with virulence, and correlates in animals with the possession of a large plasmid and expression of the plasmid-encoded, surface-expressed lipoprotein, VapA. All strains of R. equi isolated from horses with clin. disease possess a large plasmid and express VapA antigens. In addn., bacterial clearance and granuloma development in mice is linked to plasmid possession and VapA expression. Plasmid contg. strains replicate within the tissues of the mouse, whereas plasmid-cured strains are rapidly cleared. At present, the function of the VapA protein is unknown. In contrast to what is obsd. in the foal, only a small percentage of R. equi strains isolated from humans with rhodococcal disease express VapA antigens, although a high proportion of others express a related protein which is assocd. with reduced virulence and is also plasmid-encoded. In a limited no. of plasmid-neg.

human isolates, virulence has been linked to .beta.-lactam resistance, and preliminary evidence suggests that the phenotype may be phage encoded. It is likely that the immune status of the patient can influence whether a particular strain of R. equi is able to produce clin. disease, and certainly exptl. infection in mice has confirmed that an intact cellular immune response is necessary for clearance of the organism.

L246 ANSWER 39 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 97216562 EMBASE
DOCUMENT NUMBER: 1997216562
TITLE: Hydrogen sulphide produces diminished fatty acid oxidation in the rat colon in vivo: Implications for **ulcerative colitis**.
AUTHOR: Moore J.W.E.; Millard S.; Babidge W.; Rowland R.; Roediger W.E.W.
CORPORATE SOURCE: J.W.E. Moore, Gastrointestinal Services, Royal Adelaide Hospital, North Terrace, Adelaide, SA 5000, Australia
SOURCE: Australian and New Zealand Journal of Surgery, (1997) 67/5 (245-249).
Refs: 18
ISSN: 0004-8682 CODEN: ANZJA7
COUNTRY: Australia
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 009 Surgery
029 Clinical Biochemistry
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Background: Several lines of evidence suggest a possible role for reduced forms of sulphur (including sulphide) in **ulcerative colitis**. The aims of this study were to assess the metabolic profile of colonic epithelial cells after treatment in vivo with hydrogen sulphide and correlate this with mucosal histological appearances. Methods: Adult Sprague-Dawley rats had antegrade Roux-en-Y colostomies fashioned to allow access to the 'in-flow' bowel. Animals were treated with 2 mL sodium hydrosulphide (10, 20, 30 mmol/L) or saline control twice daily via the stoma for four (acute experiments) and 90 (chronic experiments) days. Isolated colonic epithelial cell suspensions prepared from such animals were incubated in the presence of [1-14C]-labelled n-**butyrate** (5 mmol/L) or [6-14C]glucose (5 mmol/L). Metabolic performance was measured radiometrically (14CO2 production) and enzymatically (ketone body production and lactogenesis). The histological appearances of treated mucosa were scored for acute inflammatory changes. Results: There was a highly significant reduction in 14CO2 production from both n-**butyrate** and glucose in all groups compared to the control in both acute and chronic experiments. There was no difference between groups with respect to histological appearance and no evidence of acute inflammation in any specimen. Conclusions: Sodium hydrosulphide impairs rat colonic epithelial metabolic performance in vivo, but does not produce mucosal inflammation.

L246 ANSWER 40 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1997:83165 CAPLUS
DOCUMENT NUMBER: 126:113013
TITLE: Antagonistic effects of sulfide and **butyrate** on proliferation of colonic mucosa. A potential role for these agents in the pathogenesis of **ulcerative colitis**
AUTHOR(S): Christl, Stefan U.; Eisner, Hans-Dieter; Dusel, Gerda; Kasper, Heinrich; Scheppach, Wolfgang

CORPORATE SOURCE: Department of Medicine, University of Wurzburg, Wurzburg, D-97080, Germany
SOURCE: Dig. Dis. Sci. (1996), 41(12), 2477-2481
CODEN: DDSCDJ; ISSN: 0163-2116
PUBLISHER: Plenum
DOCUMENT TYPE: Journal
LANGUAGE: English

AB It has been shown that feces of patients with ulcerative colitis uniformly contain sulfate reducing bacteria. Sulfide produced by these bacteria interferes with butyrate-dependent energy metab. of cultured colonocytes and may be involved in the pathogenesis of ulcerative colitis. Mucosal biopsies from the sigmoid rectum of 10 patients (no cancer, polyps, inflammatory bowel disease) were incubated with either NaCl, sodium hydrogen sulfide (1 mmol/L), a combination of both sodium hydrogen sulfide and butyrate (10 mmol/L), or butyrate. Mucosal proliferation was assessed by bromodeoxyuridine labeling of cells in S-phase. Compared to NaCl, sulfide increased the labeling of the entire crypt significantly, by 19% ($p < 0.05$). This effect was due to an expansion of the proliferative zone to the upper crypt (compartments 3-5), where the increase in proliferation was 54%. Sulfide-induced hyperproliferation was reversed when samples were coincubated with sulfide and butyrate. The study shows that sodium hydrogen sulfide induces mucosal hyperproliferation. Our data support a possible role of sulfide in the pathogenesis of UC and confirm the role of butyrate in the regulation of colonic proliferation and in the treatment of UC.

L246 ANSWER 41 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.

ACCESSION NUMBER: 96173747 EMBASE
DOCUMENT NUMBER: 1996173747
TITLE: **Butyrate** enemas in experimental **colitis** and protection against large bowel cancer in a rat model.
AUTHOR: D'Argenio G.; Cosenza V.; Delle Cave M.; Iovino P.; Della Valle N.; Lombardi G.; Mazzacca G.
CORPORATE SOURCE: Facolta di Medicina, Cattedra di Gastroenterologia, Universita Federico II, Via Pansini 5,80131 Napoli, Italy
SOURCE: Gastroenterology, (1996) 110/6 (1727-1734).
ISSN: 0016-5085 CODEN: GASTAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 016 Cancer
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background and Aims: **Butyrate** is effective in experimental colitis by increasing transglutaminase activity. Because **ulcerative colitis** increases the risk of colonic neoplasia, the aim of this study was to investigate whether **butyrate** treatment reduces mucosal sensitivity to colon cancer development in rats with experimental colitis. Methods: Colon cancer was induced by azoxymethane injections in 10 rats with trinitrobenzensulfonic acid-induced colitis and 10 rats without colitis. Three additional groups of rats with colitis were treated with **butyrate**, mesalamine, and saline enemas, respectively, twice daily for 8 weeks; 1 week after colitis induction, tumors were induced. Biopsy specimens for assessment of proliferation pattern and transglutaminase activity were obtained during the latent period of cancer development. Characteristics of tumors were recorded 27 weeks after the first exposure to azoxymethane. Results: Experimental colitis enhanced carcinogenesis; **butyrate** therapy reduced both incidence and size of tumors and also affected colonic

proliferation pattern. Transglutaminase levels were restored by **butyrate** treatment in rats with colitis. Conclusions: The protective effect of **butyrate** against large bowel cancer in experimental colitis suggests its usefulness in long-term therapy to decrease disease relapses and to reduce colon cancer risk in **ulcerative colitis**.

L246 ANSWER 42 OF 120 MEDLINE DUPLICATE 2
ACCESSION NUMBER: 97054791 MEDLINE
DOCUMENT NUMBER: 97054791 PubMed ID: 8899080
TITLE: Treatment of left-sided ulcerative **colitis** with butyrate enemas: a controlled trial.
AUTHOR: Steinhart A H; Hiruki T; Brzezinski A; Baker J P
CORPORATE SOURCE: Department of Medicine, University of Toronto, Canada.
SOURCE: ALIMENTARY PHARMACOLOGY AND THERAPEUTICS, (1996 Oct) 10 (5) 729-36.
Journal code: A5D; 8707234. ISSN: 0269-2813.
PUB. COUNTRY: ENGLAND: United Kingdom
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(MULTICENTER STUDY)
(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970227
Last Updated on STN: 19990129
Entered Medline: 19970213

AB BACKGROUND: The colonic mucosa is highly dependent upon the presence of luminal nutrients. This dependence is most marked in the distal colon. The major luminal nutrients are short chain fatty acids that are produced as a by-product of colonic fermentation of carbohydrates. Butyrate appears to be the short chain fatty acid most avidly metabolized by the colonic mucosa. It has been suggested that ulcerative colitis is, at least in part, related to an energy deficiency state of the colonic mucosa which may be secondary to impaired short chain fatty acid production, uptake or utilization. The objective of this study was to determine if butyrate given as enema therapy is effective in the treatment of active distal ulcerative colitis. METHODS: Thirty-eight patients with distal ulcerative colitis were randomly assigned to receive nightly butyrate (n = 19) or saline/placebo (n = 19) enemas. Butyrate enemas consisted of 60 mL of 80 mM sodium butyrate titrated to a pH of 7.0. Patients were assessed clinically and endoscopically at baseline and at 3 and 6 weeks follow-up. Pre- and post-treatment mucosal biopsies were assessed histologically. Response to therapy was determined by changes in a 12-point clinical disease activity index score based on patient symptoms, endoscopic mucosal appearance and physicians' global assessment. RESULTS: Clinical improvement was noted in seven of 19 (37%) butyrate-treated patients and nine of 19 (47%) placebo-treated patients (P = 0.51). Clinical remission was achieved in three patients in each group (16%). No toxicity was observed in either treatment arm. CONCLUSIONS: The results suggests that once nightly 60 mL butyrate enemas (80 mmol/L) are not efficacious in the treatment of distal ulcerative colitis.

L246 ANSWER 43 OF 120 MEDLINE
ACCESSION NUMBER: 97108216 MEDLINE
DOCUMENT NUMBER: 97108216 PubMed ID: 8950830
TITLE: The serum neutralizing antibody response in cattle to **Fusobacterium** necrophorum leukotoxoid and possible protection against experimentally induced hepatic

abscesses.
AUTHOR: Saginala S; Nagaraja T G; Tan Z L; Lechtenberg K F; Chengappa M M; Hine P M
CORPORATE SOURCE: Department of Animal Sciences, Kansas State University, Manhattan, USA.
SOURCE: VETERINARY RESEARCH COMMUNICATIONS, (1996) 20 (6) 493-504. Journal code: XCD; 8100520. ISSN: 0165-7380.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199703
ENTRY DATE: Entered STN: 19970327
Last Updated on STN: 19970327
Entered Medline: 19970317

AB The serum antileukotoxin antibody response and protection against subsequent experimental challenge with Fusobacterium necrophorum were investigated in 30 steers vaccinated with crude F. necrophorum leukotoxoid. Culture supernatant of F. necrophorum, strain 25, containing leukotoxoid was concentrated. The steers were assigned randomly to six groups (n = 5): PBS control with Stimulon adjuvant; vaccinated with concentrated supernatant diluted to provide 2.5, 5.0, 10.0, or 20.0 ml with the water-soluble Stimulon adjuvant; and 5.0 ml with the Ribi oil-emulsion adjuvant. The steers were injected subcutaneously on days 0 and 21. Blood samples were collected at weekly intervals to monitor serum antileukotoxin antibody titres. On day 42, all the steers were challenged intraportally with F. necrophorum culture. Three weeks later (day 63), the steers were killed and necropsied for examination of their livers and assessment of protection. Steers vaccinated with crude leukotoxoid tended to have higher antileukotoxin titres than the controls, but the difference was not significant. Also, the antibody titre did not appear to be dose-dependent. In the control group, 3 out of 5 steers developed liver abscesses. The incidence of liver abscesses in steers vaccinated with Stimulon adjuvant was not dose related; however, only 8 of the 25 vaccinated steers developed abscesses. None of the steers vaccinated with the 5.0 ml dose with Ribi had any abscesses. Evidence for a relationship between antileukotoxin antibody and protection was shown by the lower titre in those steers that developed abscesses compared to those that did not. It was concluded that antileukotoxin antibody titres probably provided some degree of protection against experimentally induced liver abscesses, but further dose-titration studies using Ribi or possibly another more effective adjuvant will be needed to confirm this.

L246 ANSWER 44 OF 120 MEDLINE
ACCESSION NUMBER: 96283711 MEDLINE
DOCUMENT NUMBER: 96283711 PubMed ID: 8712511
TITLE: Serum neutralizing antibody response and protection against experimentally induced liver abscesses in steers vaccinated with **Fusobacterium** necrophorum.
AUTHOR: Saginala S; Nagaraja T G; Tan Z L; Lechtenberg K F; Chengappa M M; Kemp K E; Hine P M
CORPORATE SOURCE: Department of Animal Sciences, Kansas State University, Manhattan 66506, USA.
SOURCE: AMERICAN JOURNAL OF VETERINARY RESEARCH, (1996 Apr) 57 (4) 483-8.
Journal code: 40C; 0375011. ISSN: 0002-9645.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199609
ENTRY DATE: Entered STN: 19960919
Last Updated on STN: 19970203
Entered Medline: 19960909

AB OBJECTIVE--To determine the efficacy of leukotoxin-based Fusobacterium necrophorum vaccines and dietary tylosin in providing protection against experimentally induced hepatic abscesses in steers. DESIGN--30 steers assigned randomly to 6 treatment groups of 5 steers each: 1, phosphate-buffered saline solution (PBSS; control); 2, PBSS control, fed tylosin (100 mg/steer) daily; 3, inactivated whole-cell culture with oil emulsion adjuvant; 4, culture supernatant (crude toxoid) with oil emulsion adjuvant; 5, semipurified leukotoxoid with oil emulsion adjuvant; and 6, semipurified leukotoxoid with saponin adjuvant. PROCEDURE--Steers were inoculated SC with emulsified antigen or PBSS on days 0 and 21. Blood samples were collected at weekly intervals to monitor serum antileukotoxin antibody titer. On day 42, all steers were challenge exposed intraportally with F necrophorum culture. Three weeks later (day 63), steers were euthanatized and necropsied to examine liver and assess protection. RESULTS--Antileukotoxin antibody titers of all vaccinated groups markedly increased from baseline values, and mean titers of vaccinated groups were higher than those of the control and tylosin-treated groups. Steers vaccinated with culture supernatant with oil emulsion adjuvant or semipurified leukotoxoid with saponin adjuvant had the highest mean antibody titers. All 5 steers in the control group developed liver abscesses. Tylosin feeding did not protect steers challenge exposed with F necrophorum intraportally. CONCLUSIONS--Culture supernatant was more protective than whole-cell culture or semipurified leukotoxin against experimentally induced hepatic abscesses. Partial purification of leukotoxin appeared to reduce its protective immunity.

L246 ANSWER 45 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1997:196166 BIOSIS
DOCUMENT NUMBER: PREV199799495369
TITLE: **Fusobacterium** bacteremia: Classical and atypical presentations of an uncommon disease.
AUTHOR(S): Munoz, P.; Gijon, P.; Pelaez, T.; Garrote, F.; Rodriguez-Creixems, M.; Bouza, E.
SOURCE: Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy, (1996) Vol. 36, No. 0, pp. 224. Meeting Info.: 36th ICAAC (International Conference of Antimicrobial Agents and Chemotherapy) New Orleans, Louisiana, USA September 15-18, 1996
DOCUMENT TYPE: Conference; Abstract; Conference
LANGUAGE: English

L246 ANSWER 46 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1996:475663 BIOSIS
DOCUMENT NUMBER: PREV199699205219
TITLE: Penicillin-binding proteins in **Fusobacterium** species.
AUTHOR(S): Tuner, K. (1); Wexler, H. M.; Reeves, D.; Finegold, S. M.
CORPORATE SOURCE: (1) Dep. Immunol. Microbiol. Pathol. Infect. Dis., Karolinska Inst., Huddinge Univ. Hosp., S-141 86, Huddinge Sweden
SOURCE: Anaerobe, (1996) Vol. 2, No. 3, pp. 155-162. ISSN: 1075-9964.
DOCUMENT TYPE: Article
LANGUAGE: English

AB PBPs were identified in four species of Fusobacterium. Each species had a distinctive PBP pattern, although some intra-species variation was noted.

Most species had five or six PBPs, ranging in molecular weight from apprx 100 kDa to apprx 40 kDa. The two strains of F. nucleatum tested had characteristic "wavy" PBP patterns. F. mortiferum was distinctive in possessing a very major band or complex at the PBP-2 position, whereas **F. varium** and F. necrophorum had only minor or average bands. The antibiotics tested had varying affinities for the different PBPs and distinctive morphological changes were seen upon exposure of the organisms to certain beta-lactam agents. Cefotaxime, which caused elongation in strains of two species, had greater affinity for PBPs 1 and 4 than for the other PBPs in those strains. Aztreonam, which caused elongation in **F. varium**, also had affinity for PBP-4 in that strain.

L246 ANSWER 47 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1996:510296 BIOSIS
DOCUMENT NUMBER: PREV199699232652
TITLE: Cytochemical characterization of members of the genus **Fusobacterium** by cellular carbohydrate fingerprints and selected phenotypic features.
AUTHOR(S): Heisek, Andreas; Mutters, Reinier
CORPORATE SOURCE: Inst. Med. Microbiol., Philipps Univ., Pilgrimstein 2, D-35037 Marburg Germany
SOURCE: Anaerobe, (1996) Vol. 2, No. 1, pp. 47-56.
ISSN: 1075-9964.
DOCUMENT TYPE: Article
LANGUAGE: English
AB Members of the genus Fusobacterium are associated with several human diseases especially with periodontitis and gingivitis. Unfortunately, most of the current identification methods are insufficient for the correct identification of all the species in the genus, which makes it difficult to determine the etiological role of these bacteria in periodontal diseases. Chemotaxonomical markers for precise species description are still missing. The aim of our study was to investigate the applicability of cellular carbohydrate analysis for chemotaxonomic and diagnostic purposes. Forty strains of Fusobacterium and one strain of Eubacterium plauti (formerly Fusobacterium plauti), as well as five strains of Bacteroides fragilis isolated from humans and animals were characterized by cytochemical features. The cellular carbohydrate profiles established by gas-liquid chromatography and mass spectrometry of the peracetylated aldononitriles and peracetylated methyloximes exhibited characteristic patterns suitable for the differentiation of four major groups within the genus. For further differentiation on the species level we used selected reactions of commercial test systems (API-ZYM and Rapid ID-32A) beside the carbohydrate analysis. Thus, we propose the use of cellular carbohydrate profiles in combination with selected phenotypic features for the identification of fusobacteria.

L246 ANSWER 48 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 96185268 EMBASE
DOCUMENT NUMBER: 1996185268
TITLE: Invasion of tissue culture cells by diarrhoeagenic strains of Escherichia coli which lack the enteroinvasive inv gene.
AUTHOR: Geyid A.; Fletcher J.; Gashe B.A.; Ljungh A.
CORPORATE SOURCE: Department of Medical Microbiology, University of Lund, Solvegatan 23,S-223 62 Lund, Sweden
SOURCE: FEMS Immunology and Medical Microbiology, (1996) 14/1 (15-24).
ISSN: 0928-8244 CODEN: FIMIEV
COUNTRY: Netherlands
DOCUMENT TYPE: Journal; Article

FILE SEGMENT: 004 Microbiology
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Invasive Escherichia coli strains of certain serotypes invade by the same mechanism as the Shigella sp. It has been proposed that invasion of epithelial cells by EPEC strains may also occur; this is a previously overlooked property. In the present study E. coli strains isolated from patients with diarrhoea or **ulcerative colitis**, lacking the inv plasmid mediating classical invasion, but hybridizing with probes for different adhesins, were analyzed for their ability to invade HeLa and Caco-2 cells. The majority of strains invaded Caco-2 cells to a higher extent than HeLa cells. Adhesion to Caco-2 cells was a prerequisite for subsequent invasion of the cells but EAF, eae, EAgg and other known virulence factors were not sufficient to mediate invasion. In 8/9 E. coli strains invasion was enhanced after growth under iron restriction. Growth during anaerobic conditions did not influence subsequent invasion by E. coli strains whereas 6/9 strains had their invasive ability significantly decreased after growth in the presence of 1% glucose. The invasive process was inhibited by mannose but not by lactose, fucose or galactose. Our data indicate that strains of E. coli may invade Caco-2 cells by novel mechanisms which require adhesion to the cells but which differ from those of Salmonella sp., Yersinia sp., Shigella sp. and classical enteroinvasive E. coli.

L246 ANSWER 49 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1995:281327 BIOSIS
DOCUMENT NUMBER: PREV199598295627
TITLE: Inflammatory macrophages but not resident macrophages express membrane-bound CD14, a receptor for **lipopolysaccharide**, in a mouse **model** of **ulcerative colitis**.
AUTHOR(S): Wozniak, A.; Van De Pol, E.; Doe, W. F.
CORPORATE SOURCE: Div. Clinical Sci., John Curtin Sch. Med. Res., Aust. Natl. Univ., Canberra, ACT Australia
SOURCE: Gastroenterology, (1995) Vol. 108, No. 4 SUPPL., pp. A944. Meeting Info.: 95th Annual Meeting of the American Gastroenterological Association and Digestive Disease Week San Diego, California, USA May 14-17, 1995 ISSN: 0016-5085.
DOCUMENT TYPE: Conference
LANGUAGE: English

L246 ANSWER 50 OF 120 MEDLINE
ACCESSION NUMBER: 95340186 MEDLINE
DOCUMENT NUMBER: 95340186 PubMed ID: 7615274
TITLE: Colonic epithelium is diffusely abnormal in ulcerative **colitis** and colorectal cancer.
AUTHOR: Gibson P; Rosella O; Nov R; Young G
CORPORATE SOURCE: University of Melbourne, Department of Medicine, Royal Melbourne Hospital, Victoria, Australia.
SOURCE: GUT, (1995 Jun) 36 (6) 857-63. Journal code: FVT; 2985108R. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199508
ENTRY DATE: Entered STN: 19950905 Last Updated on STN: 19990129

Entered Medline: 19950824

AB The hypothesis that the colonic epithelium is diffusely abnormal in ulcerative colitis was examined by comparing disease related responses in expression of markers of differentiation by colonic crypt cells to culture with and without butyrate. Cells were isolated from patients with normal colon (15), cancer (24), ulcerative colitis (19), or Crohn's disease (16). Alkaline phosphatase activities were measured in cell homogenates and the rate of glycoprotein synthesis assessed at the end of 24 hours of culture and expressed relative to the rate of protein synthesis as the G:P ratio. Alkaline phosphatase activities, but not G:P ratios, differed across the groups before and after 24 hour culture ($p < 0.05$), activities being lowest in the cancer group and highest in inflammatory bowel disease groups. Butyrate (1 mM) suppressed alkaline phosphatase activities in the cancer group by mean (SEM) of 17 (4) ($p = 0.006$) compared with no change in the other groups. Butyrate suppressed G:P ratios only in the cancer (6 (3)%, $p = 0.03$) and ulcerative colitis groups (5 (3)%, $p = 0.04$) and the changes in both were different ($p < 0.05$) from those in normal cells (increase of 10 (7)%). Changes in ulcerative colitis were different from those in Crohn's disease ($p = 0.029$). Responses were independent of the presence or absence of mucosal inflammation. These data confirm the diffuse nature of epithelial abnormalities in colorectal cancer. In ulcerative colitis, a different pattern of abnormality occurs, supporting the notion that the epithelium is also diffusely abnormal independent of mucosal inflammation.

L246 ANSWER 51 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1995:939916 CAPLUS
DOCUMENT NUMBER: 124:44456
TITLE: **Butyrate** and **ulcerative colitis**. Contents.
AUTHOR(S): Flourie, Bernard; Descos, Louis; Rambaud, Jean-Claude
CORPORATE SOURCE: Service d'Hepato-Gastroenterologie, Hopital Saint-Lazare, Paris, F-75010, Fr.
SOURCE: Gastroenterol. Clin. Biol. (1995), 19(6-7), 619-24
CODEN: GCBIDC; ISSN: 0399-8320
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English

AB A review, with 51 refs. including discussion on butyrate as the main energy source for colonocytes, butyrate and ulcerative colitis, the therapeutic effects of butyrate on ulcerative colitis, and the mechanism of action of butyrate.

L246 ANSWER 52 OF 120 MEDLINE
ACCESSION NUMBER: 95387723 MEDLINE
DOCUMENT NUMBER: 95387723 PubMed ID: 7658775
TITLE: The rectal approach to treatment in distal ulcerative **colitis**.
AUTHOR: Anderson F H
CORPORATE SOURCE: Division of Gastroenterology, Vancouver Hospital and Health Sciences Centre, BC, Canada.
SOURCE: LANCET, (1995 Aug 26) 346 (8974) 520-1.
Journal code: L0S; 2985213R. ISSN: 0140-6736.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199510
ENTRY DATE: Entered STN: 19951013
Last Updated on STN: 19990129
Entered Medline: 19951003

L246 ANSWER 53 OF 120 MEDLINE
ACCESSION NUMBER: 95074384 MEDLINE
DOCUMENT NUMBER: 95074384 PubMed ID: 7983262
TITLE: Chemostat flow cell system: an in vitro model for the evaluation of antiplaque agents.
AUTHOR: Herles S; Olsen S; Afflitto J; Gaffar A
CORPORATE SOURCE: Colgate Palmolive, Piscataway, New Jersey 08854.
SOURCE: JOURNAL OF DENTAL RESEARCH, (1994 Nov) 73 (11) 1748-55. Journal code: HYV; 0354343. ISSN: 0022-0345.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 199501
ENTRY DATE: Entered STN: 19950116
Last Updated on STN: 19970203
Entered Medline: 19950104

AB We developed an experimental in vitro model of dental plaque to assess the potential efficacy of antiplaque agents. The model used a chemostat, which provided a continuous source of 5 species of oral bacteria grown in an artificial "saliva-like" medium. This mixture was pumped through six flow cells, each containing two types of surfaces on which plaque formed and was subsequently measured. Formation of bacterial plaque on hydroxyapatite surfaces was assessed by measurement of the DNA and protein content of the plaque film. The amount of bacterial plaque formed on germanium surfaces was measured by attenuated total reflectance (ATR/FT-IR) spectroscopy. Plaque viability was also assessed by a fluorescent staining technique. The quantity of plaque formed on both types of surfaces gradually increased with the duration of flow (from 24 to 72 h) through the cells during a 72-hour experimental period. The flow cells were then pulsed with experimental treatment solutions for 30 s, twice daily. Parallel to results of human clinical studies, the model was capable of discriminating among water, a placebo mouthrinse, and an active antimicrobial mouthrinse formulation containing 0.03% triclosan. It therefore offers a valuable alternative to animal model testing and allows for more rapid evaluations under well-controlled experimental conditions.

L246 ANSWER 54 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 94047716 EMBASE
DOCUMENT NUMBER: 1994047716
TITLE: **Butyrate**, mesalamine, and factor XIII in experimental **colitis** in the rat: Effects on transglutaminase activity.
AUTHOR: D'Argenio G.; Cosenza V.; Sorrentini I.; De Ritis F.; Gatto A.; Cave M.D.; D'Armiento F.P.; Mazzacca G.
CORPORATE SOURCE: Cattedra di Gastroenterologia, Facolta di Medicina, Universita 'Federico II', Via Pansini 5,80131 Naples, Italy
SOURCE: Gastroenterology, (1994) 106/2 (399-404). ISSN: 0016-5085 CODEN: GASTAB
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
037 Drug Literature Index
048 Gastroenterology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Background/Aims: **Butyrate** and factor XIII may improve **ulcerative colitis**; they also affect tissue and serum transglutaminase levels. We investigated the therapeutic potential of

sodium **butyrate** and factor XIII and the role of transglutaminase during mucosal repair in experimental colitis. Methods: Rats with induced colitis were treated with sodium **butyrate**, mesalamine, sodium **butyrate** plus mesalamine, or saline enemas. Thromboxane B2 was monitored as index of inflammation. In a fifth group, the effectiveness of intravenous Factor XIII was assessed. Results: Sodium **butyrate**, alone or plus mesalamine, reduced histological activity from 13.7 .+-. 1.7 (saline) to 2.5 .+-. 1.3 and 2.3 .+-. 1.1 ($P < 0.01$), respectively. Transglutaminase, reduced in the colons of the saline group (783 .+-. 157 vs. normal 1800 .+-. 192 mU/g; $P < 0.01$), returned toward normal values in the sodium **butyrate** or sodium **butyrate** plus mesalamine groups (1390 .+-. 228 and 1226 .+-. 172 mU/g, respectively; $P < 0.01$ vs. saline). Furthermore, sodium **butyrate** plus mesalamine reduced thromboxane B2 levels by day 5 (0.92 .+-. 0.16 vs. saline 1.85 .+-. 0.34 ng/mL; $P < 0.05$). Factor XIII therapy improved the histological picture (2.7 .+-. 2.1 vs. saline 13.8 .+-. 1.7; $P < 0.01$) and increased transglutaminase levels both in serum (2.81 .+-. 0.11 vs. saline 1.45 .+-. 0.09 mU/mL; $P < 0.01$) and in colon (1503 .+-. 127 vs. saline 747 .+-. 103). Conclusions: Sodium **butyrate** and factor XIII improve colitis, sodium **butyrate** plus mesalamine reduce early thromboxane B2 synthesis, and transglutaminase(s) plays a role in ulcer healing.

L246 ANSWER 55 OF 120 MEDLINE
ACCESSION NUMBER: 94315893 MEDLINE
DOCUMENT NUMBER: 94315893 PubMed ID: 8041140
TITLE: Butyrate increases colonocyte protein synthesis in ulcerative **colitis**.
AUTHOR: Frankel W; Lew J; Su B; Bain A; Klurfeld D; Einhorn E; MacDermott R P; Rombeau J
CORPORATE SOURCE: Department of Surgery, Hospital of the University of Pennsylvania, Philadelphia 19104.
CONTRACT NUMBER: 5-T32-CA 09430-10 (NCI)
SOURCE: JOURNAL OF SURGICAL RESEARCH, (1994 Jul) 57 (1) 210-4. Journal code: K7B; 0376340. ISSN: 0022-4804.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199408
ENTRY DATE: Entered STN: 19940905
Last Updated on STN: 19990129
Entered Medline: 19940822

AB Butyrate promotes epithelial cell healing and improves symptoms when administered rectally in patients with distal ulcerative colitis (UC). It was hypothesized that butyrate may enhance healing in patients with UC by stimulating colonocyte proliferation and/or protein production. Mucosa from the descending colon was obtained from patients with UC (n = 5), Crohn's disease (n = 8), diverticulitis (n = 6), and cancer (normal tissue 10 cm from tumor; n = 10). Epithelial cells were isolated using dispase/collagenase and differential sedimentation and incubated for 4 hr at 37 degrees C with either Na butyrate (10 mM) or NaCl (10 mM). Protein synthesis was assessed by [14C]leucine incorporation and proliferation was determined with [3H]thymidine. Mean viability and purity were >88%. Spontaneous proliferation was significantly increased in UC when compared to diverticulitis and normal controls. Butyrate significantly increased protein synthesis in UC epithelial cells when compared to saline control. The therapeutic effects of butyrate in patients with UC may be due to its use by epithelial cells as a metabolic fuel to increase protein production and promote healing.

L246 ANSWER 56 OF 120 LIFESCI COPYRIGHT 2002 CSA
ACCESSION NUMBER: 95:119892 LIFESCI
TITLE: **Fusobacterial** infections in children
AUTHOR: Brook, I.
CORPORATE SOURCE: P.O. Box 70412, Chevy Chase, MD 20813-0412, USA
SOURCE: J. INFECT., (1994) vol. 28, no. 2, pp. 155-165.
ISSN: 0163-4453.
DOCUMENT TYPE: Journal
FILE SEGMENT: J
LANGUAGE: English
SUMMARY LANGUAGE: English
AB A total of 243 strains of Fusobacteria species was recovered from 226 of 1399 (16%) specimens obtained from 213 children. The strains included 65 (27%) Fusobacterium sp., 144 (59%) Fusobacterium nucleatum, 25 (10%) Fusobacterium necrophorum, five (2%) **Fusobacterium varium**, three (1%) Fusobacterium mortiferum, and one (0.4%) Fusobacterium gonidiaformans. Most Fusobacteria species were recovered from patients with abscesses (100), aspiration pneumonia (24), paronychia (15), bites (14), chronic sinusitis (ten), chronic otitis media (nine), and osteomyelitis (eight). Predisposing conditions were noted in 32 (15%) of the cases. These included immunodeficiency in nine (4%), steroid therapy in eight (4%), previous surgery in six (3%), diabetes in six (3%) and malignant neoplasms in five (2%). Fusobacteria sp. was the only isolate in 16 (8%) instances while mixed infections were encountered in 197 (92%) patients. The organisms most commonly isolated with Fusobacteria sp. were anaerobic cocci (155), pigmented Prevotella sp. and Porphyromonas sp. (95), Bacteroides fragilis group (80), Escherichia coli (43) and Bacteroides sp. (39). Most strains of B. fragilis group and E. coli were recovered from intra-abdominal infections and skin and soft tissue infections proximal to the rectal area. Most pigmented Prevotella sp. and Porphyromonas sp. were recovered from oropharyngeal and pulmonary sites and from sites around the head and neck. **Antimicrobial** therapy was administered to all patients; surgical drainage was performed in 85 (40%). All patients, except two who died, recovered. These findings illustrate the prevalence of Fusobacteria sp. associated with infections in children.

L246 ANSWER 57 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1995:253592 BIOSIS
DOCUMENT NUMBER: PREV199598267892
TITLE: Role of Immune Networks in Inflammatory Bowel Disease (IBD) with Special Reference to **Ulcerative Colitis** and Crohn's Disease: A Proposal for a New Pathogenic **Model** of IBD.
AUTHOR(S): Jirillo, E. (1); Greco, B. (1); Caradonna, L. (1); Rembouskos, G.; Di Leo, A.; Giorgio, I.
CORPORATE SOURCE: (1) Ist. Microbiol. Med., Univ. degli Studi Bari, Bari Italy
SOURCE: EOS-Rivista di Immunologia ed Immunofarmacologia, (1994) Vol. 14, No. 3-4, pp. 121-131.
ISSN: 0392-6699.
DOCUMENT TYPE: General Review
LANGUAGE: English
SUMMARY LANGUAGE: English; Italian
AB Inflammatory bowel disease (IBD) comprise two main pathological conditions known as ulcerative colitis (UC) and Crohn's disease (CD). Despite many efforts to clarify some possible aetiologic factors of IBD no final conclusions have been achieved by several groups of investigators interested in this field. In particular, conflicting results are emerging

from studies on the role of cytokines (CKs) and other related mediators produced in the course of IBD. The major scope of the present article is to provide an overview on the immune mechanisms involved in IBD and, at the same time, to propose a new model of pathogenesis for both UC and CD. In this respect, our opinion is that bacterial **lipopolysaccharides** from gram-negative bacteria represent potent triggers of mediator release in IBD, thus implying that different compartments of the immune system are involved. If this hypothesis will be proved in future, possible novel diagnostic and therapeutic approaches for the treatment of IBD should be taken into serious account.

L246 ANSWER 58 OF 120 MEDLINE
ACCESSION NUMBER: 94140218 MEDLINE
DOCUMENT NUMBER: 94140218 PubMed ID: 8307454
TITLE: Butyrate oxidation is impaired in the colonic mucosa of sufferers of quiescent ulcerative **colitis**.
AUTHOR: Chapman M A; Grahn M F; Boyle M A; Hutton M; Rogers J; Williams N S
CORPORATE SOURCE: Academic Department of Surgery, London Hospital Medical College.
SOURCE: GUT, (1994 Jan) 35 (1) 73-6.
Journal code: FVT; 2985108R. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199403
ENTRY DATE: Entered STN: 19940330
Last Updated on STN: 19990129
Entered Medline: 19940315

AB The short chain fatty acids, acetate, propionate, and butyrate are produced by colonic bacterial fermentation of non-starch polysaccharides. Butyrate is the major fuel source for the colonic epithelium and there is evidence to suggest that its oxidation is impaired in ulcerative colitis. Triplicate biopsy specimens were taken at colonoscopy from five regions of the large bowel in 15 sufferers of ulcerative colitis. These patients all had mild or quiescent colitis as assessed by clinical condition, mucosal endoscopic and histological appearance. The rate of oxidation of glucose, glutamine, and butyrate through to carbon dioxide was compared with that in biopsy specimens from 28 patients who had no mucosal abnormality. Butyrate (272 (199-368)) was the preferred fuel source for the colitic mucosa followed by glutamine (33 (24-62)) then glucose (7.2 (5.3-15)) pmol/micrograms/hour; medians and 95% confidence intervals, $p < 0.01$. There was no regional difference in the rate of utilisation of these metabolites. In the group with colitis the rate of butyrate oxidation to carbon dioxide was significantly impaired compared with that in normal mucosa decreasing from 472 (351-637) pmol/micrograms/hour to 272 (199-368) pmol/micrograms/hour; median and 95% confidence intervals, $p = 0.016$. The rate of glucose and glutamine utilisation were not significantly different between normal and colitic mucosa. These data confirm that in quiescent ulcerative colitis there is an impairment of butyrate oxidation.

L246 ANSWER 59 OF 120 MEDLINE
ACCESSION NUMBER: 94063651 MEDLINE
DOCUMENT NUMBER: 94063651 PubMed ID: 8244158
TITLE: Colonic epithelial metabolism in ulcerative **colitis**
.
COMMENT: Comment on: Gut. 1993 Nov;34(11):1552-8
AUTHOR: Roediger W E
SOURCE: GUT, (1993 Nov) 34 (11) 1646.

Journal code: FVT; 2985108R. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom
Commentary
Letter
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940201
Last Updated on STN: 19990129
Entered Medline: 19931223

L246 ANSWER 60 OF 120 MEDLINE
ACCESSION NUMBER: 94063630 MEDLINE
DOCUMENT NUMBER: 94063630 PubMed ID: 8244143
TITLE: Ileal and colonic epithelial metabolism in quiescent ulcerative **colitis**: increased glutamine metabolism in distal colon but no defect in butyrate metabolism.
COMMENT: Comment in: Gut. 1993 Nov;34(11):1646
Comment in: Gut. 1994 Aug;35(8):1152-3
Erratum in: Gut 1994 Aug;35(8):1154
AUTHOR: Finnie I A; Taylor B A; Rhodes J M
CORPORATE SOURCE: Department of Medicine, University of Liverpool.
SOURCE: GUT, (1993 Nov) 34 (11) 1552-8.
Journal code: FVT; 2985108R. ISSN: 0017-5749.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199312
ENTRY DATE: Entered STN: 19940201
Last Updated on STN: 19990129
Entered Medline: 19931223

AB Previous studies have shown that butyrate is an important energy source for the distal colon, and that its metabolism may be defective in ulcerative colitis (UC). A similar metabolic defect in the ileum might account for the occurrence of 'pouchitis' in UC patients after colectomy. A method has been developed that allows the measurement of metabolism in ileocolonoscopic biopsy specimens, and this has been used to assess butyrate and glutamine metabolism in quiescent UC and controls. Preliminary experiments showed optimal metabolism of butyrate at 1 mmol/l. In controls glutamine metabolism was greater in the ascending (mean (SD)) (4.9 (3.2) nmol/h/micrograms protein) than in the descending colon (1.4 0.7)) ($p < 0.05$, Mann-Whitney U test), but butyrate metabolism was similar in the two regions (ascending 62.6 (44.2), descending 51.5 (32.0)). Consequently ratios of butyrate/glutamine metabolism were higher in the descending colon (20.6 (14.3)) than in the ascending colon (14.3 (9.6)) ($p < 0.05$). In UC, rates of butyrate metabolism were similar in the ascending (92.5 (58.3) nmol/h/micrograms protein) and descending (93.3 (115)) colon, and these were not significantly different from controls. In UC, glutamine metabolism was similar in the ascending (6.2 (7.7) nmol/h/micrograms protein) and descending colon (7.8 (7.9)); the metabolism in the descending colon was significantly greater than in controls ($p < 0.01$). Butyrate (135 (56) nmol/h/microgram protein) and glutamine (24.1 (16.2)) metabolism in the ileum in UC, were not significantly different from control values (butyrate 111 (57), glutamine 15.5 (15.6)).(ABSTRACT TRUNCATED AT 250 WORDS)

L246 ANSWER 61 OF 120 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 93:138385 CABA

DOCUMENT NUMBER: 932292677
TITLE: **Fusobacterium** pseudonecrophorum is a synonym for **Fusobacterium varium**
AUTHOR: Bailey, G. D.; Love, D. N.
CORPORATE SOURCE: Department of Veterinary Pathology, University of Sydney, Sydney, NSW 2006, Australia.
SOURCE: International Journal of Systematic Bacteriology, (1993) Vol. 43, No. 4, pp. 819-821. 17 ref.
ISSN: 0020-7713
DOCUMENT TYPE: Journal
LANGUAGE: English

AB DNA-DNA hybridization by the S1 nuclease method showed that **Fusobacterium varium** ATCC 8501T had 88 and 79% DNA homology with F. pseudonecrophorum JCM 3722T and JCM 3723, respectively, While F. pseudonecrophorum JCM 3722T showed 81 and 82% DNA homology with **F. varium** ATCC 8501T and F. pseudonecrophorum JCM 3723, respectively. These genetic data and their similar phenotypic characteristics suggest that **F. varium** (Eggerth and Gagnon 1933) Moore and Holdeman 1969 and F. pseudonecrophorum (ex Prevot 1940) Shinjo et al. 1990 belong to a single species. It is proposed that strains JCM 3722 and JCM 3723 of F. pseudonecrophorum be transferred to the species **F. varium**.

L246 ANSWER 62 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 93336564 EMBASE
DOCUMENT NUMBER: 1993336564
TITLE: Sulphide impairment of substrate oxidation in rat colonocytes: A biochemical basis for **ulcerative colitis**?.
AUTHOR: Roediger W.E.W.; Duncan A.; Kapaniris O.; Millard S.
CORPORATE SOURCE: University of Adelaide, Department of Surgery, Queen Elizabeth Hospital,Woodville, SA, Australia
SOURCE: Clinical Science, (1993) 85/5 (623-627).
ISSN: 0143-5221 CODEN: CSCIAE
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 005 General Pathology and Pathological Anatomy
006 Internal Medicine
026 Immunology, Serology and Transplantation
029 Clinical Biochemistry
048 Gastroenterology
037 Drug Literature Index
LANGUAGE: English
SUMMARY LANGUAGE: English

AB Isolated colonic epithelial cells of the rat were incubated for 40 min with [6-14C]glucose and n-[1-14C]**butyrate** in the presence of 0.1-2.0 mmol/l NaHS, a concentration range found in the human colon. Metabolic products, 14CO2, acetoacetate, .beta.-hydroxybutyrate and lactate, were measured and injury to cells was judged by diminished production of metabolites. Oxidation of n-**butyrate** to CO2 and acetoacetate was reduced at 0.1 and 0.5 mmol/l NaHS, whereas glucose oxidation remained unimpaired. At 1.0-2.0 mmol/l NaHS, n-**butyrate** and glucose oxidation were dose-dependently reduced at the same rate. To bypass short-chain acyl-CoA dehydrogenase activity necessary for [illegible]-oxidation, ketogenesis from crotonate was measured in the [illegible] 1.0 mmol/l NaHS. Suppression by sulphide of ketogenesis from [illegible]-10.5 .+-. 6.1%) compared with control conditions was not [illegible], whereas suppression of ketogenesis from n-**butyrate** [illegible]. 5.14%) was significant (P = <0.01). Inhibition of FAD-linked [illegible]as more affected by NaHS than was NAD-linked oxidation.

Searched by Thom Larson, STIC, 308-7309

L-Methionine (5.0 mmol/l) significantly redressed the impaired .beta.-oxidation induced by NaHS. Methionine equally improved CO2 and ketone body production, suggesting a global reversal of the action of sulphide. Sulphide-induced oxidative changes closely mirror the impairment of .beta.-oxidation observed in colonocytes of patients with **ulcerative colitis**. A hypothesis for the disease process of **ulcerative colitis** is that sulphides may form persulphides with butyryl-CoA, which would inhibit cellular short-chain acyl-CoA dehydrogenase and .beta.-oxidation to induce an energy-deficiency state in colonocytes and mucosal inflammation.

L246 ANSWER 63 OF 120 MEDLINE
ACCESSION NUMBER: 93313085 MEDLINE
DOCUMENT NUMBER: 93313085 PubMed ID: 8391865
TITLE: Lethal photosensitization of oral anaerobic bacteria.
AUTHOR: Wilson M; Dobson J
CORPORATE SOURCE: Department of Microbiology, Institute of Dental Surgery, London, United Kingdom.
SOURCE: CLINICAL INFECTIOUS DISEASES, (1993 Jun) 16 Suppl 4 S414-5. Journal code: A4J; 9203213. ISSN: 1058-4838.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199308
ENTRY DATE: Entered STN: 19930820
Last Updated on STN: 19930820
Entered Medline: 19930812

L246 ANSWER 64 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1994:427451 BIOSIS
DOCUMENT NUMBER: PREV199497440451
TITLE: A new beta-lactamase produced by **Fusobacterium varium**.
AUTHOR(S): Nord, C. E. (1); Lindmark, A.; Persson, I.
CORPORATE SOURCE: (1) Huddinge Univ. Hosp., Karolinska Inst., Stockholm Sweden
SOURCE: Program and Abstracts of the Interscience Conference on Antimicrobial Agents and Chemotherapy, (1993) Vol. 33, No. 0, pp. 397.
Meeting Info.: 33rd Interscience Conference on Antimicrobial Agents and Chemotherapy New Orleans, Louisiana, USA October 17-20, 1993
ISSN: 0733-6373.
DOCUMENT TYPE: Conference
LANGUAGE: English

L246 ANSWER 65 OF 120 MEDLINE
ACCESSION NUMBER: 92262942 MEDLINE
DOCUMENT NUMBER: 92262942 PubMed ID: 1585083
TITLE: Further observations on the weak immunogenicity of **Fusobacterium** necrophorum.
AUTHOR: Smith G R; Wallace L M
CORPORATE SOURCE: Institute of Zoology, Zoological Society of London.
SOURCE: RESEARCH IN VETERINARY SCIENCE, (1992 Mar) 52 (2) 262-3. Journal code: R7D; 0401300. ISSN: 0034-5288.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920626
Last Updated on STN: 19920626
Entered Medline: 19920615

AB Five virulent strains of Fusobacterium necrophorum resembled a single strain examined earlier by possessing little or no immunogenicity: severe subcutaneous infections cured with metronidazole failed to increase the resistance of mice to subcutaneous challenge 22 days after the cessation of treatment.

L246 ANSWER 66 OF 120 MEDLINE
ACCESSION NUMBER: 92278223 MEDLINE
DOCUMENT NUMBER: 92278223 PubMed ID: 1593964
TITLE: Numerical taxonomy of Bacteroides and other genera of Gram-negative anaerobic rods.
AUTHOR: Jenkins S A; Drucker D B; Hillier V F; Ganguli L A
CORPORATE SOURCE: Department of Microbiology, Hope Hospital, Salford, Great Britain.
SOURCE: MICROBIOS, (1992) 69 (279) 139-54.
Journal code: MXS; 0207257. ISSN: 0026-2633.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199206
ENTRY DATE: Entered STN: 19920710
Last Updated on STN: 19920710
Entered Medline: 19920629

AB A numerical taxonomy was performed on 157 cultures (141 different strains) of species of Bacteroides, Polyphyromonas, Prevotella [not Prevotella (Labroue, 1976)] and Fusobacterium. Isolates were each tested for 111 phenotypic characters which included possession of constitutive enzymes, fermentation of specific carbohydrates, gas chromatographic analysis of metabolic end-products and of cellular carboxylic acid composition. Computation of similarity coefficients was followed by a single-linkage cluster analysis. At the 94% similarity level, the following groupings at genus level were apparent: (1) Bacteroides ureolyticus; (2) Fusobacterium mortiferum, F. necrogenes, F. necrophorum, F. nucleatum and F. **varium**; (3) B. caccae, B. distasonis, B. eggerthii, B. fragilis, B. merdae, B. ovatus, B stercoris, B. thetaiotaomicron, B. uniformus and B. vulgatus; (4) B. splanchnicus; (5) Porphyromonas asaccharolytica; (6) B. bivius (Prevotella bivia); (7) B. disiens (P. disiens); (8) B. intermedius (P. intermedia);and (9) B. melaninogenicus (P. melaninogenica). Single isolates of B. ruminicola (P. ruminicola), B. denticola (P. denticola) and B. capillosus did not cluster with other strains.

L246 ANSWER 67 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1993:205006 CAPLUS
DOCUMENT NUMBER: 118:205006
TITLE: Lipogenesis from n-**butyrate** in colonocytes. Action of reducing agent and 5-aminosalicylic acid with relevance to **ulcerative colitis**
AUTHOR(S): Roediger, W. E. W.; Kapaniris, O.; Millard, S.
CORPORATE SOURCE: Dep. Surg., Queen Elizabeth Hosp., Adelaide, 5011, Australia
SOURCE: Mol. Cell. Biochem. (1992), 118(2), 113-18
CODEN: MCBIB8; ISSN: 0300-8177
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Cell membrane of colonic epithelial cells (CEC) in ulcerative colitis show structural abnormalities which are specific to the disease and which suggest impaired lipogenesis in CECs. Lipogenesis from [1-14C]-n-butyrate, the chief oxidative fuel of colonic epithelial cells, was measured in isolated CECs under control conditions, with or without glucose and in the presence of mercaptoacetate, a major reducing agent in the colonic lumen. Glucose stimulated lipogenesis from [1-14C]-butyrate which was reversed by 5 mM mercaptoacetate. Mercaptoacetate diminished CEC thiolase activity (EC 2.3.1.9). 5-Aminosalicylic acid reversed the adverse effects of mercaptoacetate in the saponifiable fraction of extd. lipids. Changes in lipogenesis due to colonic luminal reducing agents would affect the barrier function of CECs, a feature relevant to the disease process of ulcerative colitis.

L246 ANSWER 68 OF 120 MEDLINE
ACCESSION NUMBER: 93160916 MEDLINE
DOCUMENT NUMBER: 93160916 PubMed ID: 1369193
TITLE: Sensitization of oral bacteria to killing by low-power laser radiation.
AUTHOR: Wilson M; Dobson J; Harvey W
CORPORATE SOURCE: Microbiology Laboratory, Institute of Dental Surgery, London, UK.
SOURCE: CURRENT MICROBIOLOGY, (1992 Aug) 25 (2) 77-81.
Journal code: BMW; 7808448. ISSN: 0343-8651.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: B
ENTRY MONTH: 199303
ENTRY DATE: Entered STN: 19950809
Last Updated on STN: 19950809
Entered Medline: 19930318

AB Twenty-seven compounds were screened for their ability to sensitize Streptococcus sanguis to killing by light from a 7.3-mW Helium/Neon (HeNe) laser. Bacteria were mixed with various concentrations of the test compounds, spread over the surfaces of agar plates, and then exposed to light from the HeNe laser for various time periods. The plates were then incubated and examined for zones of inhibition. Those compounds found to be effective photosensitizers were then tested against Porphyromonas gingivalis, Actinobacillus actinomycetemcomitans, and Fusobacterium nucleatum. Toluidine blue O, azure B chloride, and methylene blue at concentrations of 0.005% (wt/vol) were effective photosensitizers of all four species, enabling killing of bacteria following exposure to laser light for only 30 s.

L246 ANSWER 69 OF 120 MEDLINE
ACCESSION NUMBER: 92307334 MEDLINE
DOCUMENT NUMBER: 92307334 PubMed ID: 1612357
TITLE: Effect of butyrate enemas on the colonic mucosa in distal ulcerative **colitis**.
COMMENT: Comment in: Gastroenterology. 1992 Jul;103(1):336-8
Comment in: Gastroenterology. 1992 Nov;103(5):1709-10
AUTHOR: Scheppach W; Sommer H; Kirchner T; Paganelli G M; Bartram P; Christl S; Richter F; Dusel G; Kasper H
CORPORATE SOURCE: Department of Medicine, University of Wurzburg, Germany.
SOURCE: GASTROENTEROLOGY, (1992 Jul) 103 (1) 51-6.
Journal code: FH3; 0374630. ISSN: 0016-5085.
PUB. COUNTRY: United States
(CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)

(RANDOMIZED CONTROLLED TRIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199207
ENTRY DATE: Entered STN: 19920807
Last Updated on STN: 19990129
Entered Medline: 19920724

AB Short-chain fatty acid irrigation has been shown to ameliorate inflammation in diversion colitis. In this study the effect of butyrate enemas was tested in 10 patients with distal ulcerative colitis who had been unresponsive to or intolerant of standard therapy for 8 weeks. They were treated for 2 weeks with sodium butyrate (100 mmol/L) and 2 weeks with placebo in random order (single-blind trial). Before and after treatment, clinical symptoms were noted and the degree of inflammation was graded endoscopically and histologically. Rectal proliferation was assessed by autoradiography. After butyrate irrigation, stool frequency (n/day) decreased from 4.7 +/- 0.5 to 2.1 +/- 0.4 (P less than 0.01) and discharge of blood ceased in 9 of 10 patients. The endoscopic score fell from 6.5 +/- 0.4 to 3.8 +/- 0.8 (P less than 0.01). The histological degree of inflammation decreased from 2.4 +/- 0.3 to 1.5 +/- 0.3 (P less than 0.02). Overall crypt proliferation was unchanged, but the upper crypt-labeling index fell from 0.086 +/- 0.019 to 0.032 +/- 0.003 (P less than 0.03). On placebo, all of these parameters were unchanged. These data support the view that butyrate deficiency may play a role in the pathogenesis of distal ulcerative colitis and that butyrate irrigation ameliorates this condition.

L246 ANSWER 70 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:322876 BIOSIS
DOCUMENT NUMBER: BA92:33391
TITLE: PATHWAYS OF GLUTAMATE CATABOLISM AMONG **FUSOBACTERIUM**-SPP.
AUTHOR(S): GHARBIA S E; SHAH H N
CORPORATE SOURCE: DEP. ORAL MICROBIOL., LONDON HOSP. MED. COLL., LONDON E1 2AD, UK.
SOURCE: J GEN MICROBIOL, (1991) 137 (5), 1201-1206.
CODEN: JGMIAN. ISSN: 0022-1287.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Glutamate is a major source of energy for Fusobacterium species its mode of catabolism has not hitherto been elucidated. Cell suspensions of F. nucleatum and **F. varium**, as representative species from the oral cavity and gastrointestinal tract, respectively, both decarboxylated position-labelled glutamate but by different pathways. 14CO2 was released only from C-5 by F. nucleatum whereas **F. varium** decarboxylated glutamate at either C-1 or C-5. In both species, 2 mols of glutamate fermented yielded 2 mols of acetate and 1 mol of butyrate, suggesting possibility of three metabolic pathways: the 2-oxoglutarate, mesaconate and 4-aminobutyrate pathways. Enzymes representative of the three pathways were asssayed for in cell-free extracts of fusobacteria. All species tested possessed high levels of both glutamate dehydrogenase and 2-oxoglutarate reductase, indicating the presence of the 2-oxoglutarate pathway. Enzymes representative of the mesaconate pathway were detected in F. sulci, F. ulcerans, F. mortiferum and **F. varium**, while the latter two species also possessed the 4-aminobutyrate pathway. The pathways of glutamate catabolism therefore bore no relationship to the site of isolation of the fusobacteria tested but instead correlated with their chemotaxonomic properties. Thus, **F. varium**, F. mortiferum, F. ulcerans and F. sulci, which possess a **peptidoglycan** structure

based on diaminopimelic acid, had either two or three pathways for glutamate catabolism whereas F. nucleatum and other species that have a lanthionine-based murein metabolized glutamate solely by the 2-oxoglutarate pathway.

L246 ANSWER 71 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:353236 BIOSIS
DOCUMENT NUMBER: BR41:37751
TITLE: VARIATION IN **PEPTIDOGLYCAN** COMPOSITION OF **FUSOBACTERIUM** SPECIES.
AUTHOR(S): FRASER H Y; GHARBIA S E; SHAH H N
CORPORATE SOURCE: DEP. ORAL MICROBIOL., LONDON HOSP. MED. COLL., TURNER ST., WHITECHAPEL, LONDON E1 2AD, UK.
SOURCE: BORRIELLO, S. P. (ED.). CLINICAL AND MOLECULAR ASPECTS OF ANAEROBES; SIXTH BIENNIAL ANAEROBE DISCUSSION GROUP INTERNATIONAL SYMPOSIUM, CAMBRIDGE, ENGLAND, UK, JULY 20-22, 1989. XXII+329P. WRIGHTSON BIOMEDICAL PUBLISHING LTD.: PETERSFIELD, ENGLAND, UK. ILLUS, (1991) 0 (0), 313-314.
ISBN: 1-871816-04-1.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 72 OF 120 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 91:72398 CABA
DOCUMENT NUMBER: 912251215
TITLE: A sensitive method for isolating **Fusobacterium** necrophorum from faeces
AUTHOR: Smith, G. R.; Barton, S. A.; Wallace, L. M.
CORPORATE SOURCE: Institute of Zoology, The Zoological Society of London, Regent's Park, London NW1 4RY, UK.
SOURCE: Epidemiology and Infection, (1991) Vol. 100, No. 2, pp. 311-317. 12 ref.
ISSN: 0950-2688
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The isolation of F. necrophorum present in small numbers in heavily contaminated material such as faeces or soil is hampered by the lack of an efficient selective medium and by the high minimum infective dose of the organism. A sensitive method for the detection and isolation of faecal strains of F. necrophorum type A was based on the s.c. injection of faeces, suspended (5% w/v) in broth culture of Actinomyces (Corynebacterium) pyogenes or Staphylococcus aureus to increase **fusobacterial** infectivity, into mice pretreated with clostridial antitoxins. When necrobacillosis developed F. necrophorum was identified microscopically in tissue from the advancing edge of the lesion and isolated on a partly selective medium. The enhancement of **fusobacterial** infectivity produced by A. pyogenes and by S. aureus was high, but the latter was slightly the more efficient, enabling as few as 80 F. necrophorum organisms/g of faeces to be detected. Use of the method showed that 3 of 16 wallabies had F. necrophorum in their faeces at the time of examination. Numerous epidemiological applications are suggested.

L246 ANSWER 73 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1990:494612 CAPLUS
DOCUMENT NUMBER: 113:94612
TITLE: Nutritional factors reducing potent antagonistic effects of 5 bacterial species of predominant human

colonic flora on Salmonella typhimurium in anaerobic continuous flow cultures
AUTHOR(S): Ushijima, Tsutomu
CORPORATE SOURCE: Dep. Microbiol., Shiga Univ. Med. Sci., Otsu, 520-21, Japan
SOURCE: Igaku to Seibutsugaku (1990), 120(6), 231-5
CODEN: IGSBAL; ISSN: 0019-1604
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
AB Five dominant species in human colonic flora, Escherichia coli ATCC 25922, 11775, SU 5034, IFO 12713, Enterobactor aerogenes IFO 13534, Enterococcus faecalis IFO 12969, Bacteroides ovatus SU 39, Fusobacterium varium ATCC 8501, showed antagonism against Salmonella typhimurium LT-2, when they were incubated in anaerobic continuous flow culture using the MCM medium without acetic acid, propionic acid, **butyric** acid, lactic acid and succinic acid, and contg. 0.1% sucrose, 0.1% lactose, 0.2% starch, 0.05G glucose and 0.05% sorbose. But, this antagonism was reduced when concn. of arginine, serine, threonine or aspartic acid in the medium increased.

L246 ANSWER 74 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1991:66319 BIOSIS
DOCUMENT NUMBER: BA91:34979
TITLE: IDENTIFICATION OF **FUSOBACTERIUM**-SPP BY THE ELECTROPHORETIC MIGRATION OF GLUTAMATE DEHYDROGENASE AND 2 OXOGLUTARATE REDUCTASE IN RELATION TO THEIR DNA BASE COMPOSITION AND **PEPTIDOGLYCAN** DIBASIC AMINO ACIDS.
AUTHOR(S): GHARBIA S E; SHAH H N
CORPORATE SOURCE: DEP. ORAL MICROBIOLOGY, LONDON HOSP. MED. COLL., TURNER STREET, WHITECHAPEL, LONDON E1 2AD.
SOURCE: J MED MICROBIOL, (1990) 33 (3), 183-188.
CODEN: JMMIAV. ISSN: 0022-2615.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Rapid identification of Fusobacterium spp. is hampered by their inability to ferment carbohydrates and the availability of relatively few useful phenotypic characters. In an attempt to identify new diagnostic markers for species, we reported recently the potential utility of glutamate dehydrogenase (GDH) electrophoretic mobilities for distinguishing eight species of Fusobacterium. We have extended these observations to include all recognised members of the genus except F. prausnitzii and F. perfoetens, and our results show that they cluster into three broad electrophoretic groups. Some species, such as F. periodonticum, F. simiae and F. necrophorum possessed GDH with similar electrophoretic mobilities. However, within such clusters, the electrophoretic migration of 2-oxoglutarate reductase (OGR) distinguished between species. Neither GDH or OGR mobility alone clearly differentiated all species, but their combined use provided unambiguous discrimination of all species except **F. varium** and F. mortiferum. The DNA base compositions of all species except F. naviforme (ATCC 25832) and F. sulci, were within the range 26-34 mol% G + C, suggesting the genus may be homogeneous. However, the **peptidoglycan** composition divided the genus into two major groups that contained either lanthionine or diaminopimelic acid; F. mortiferum **peptidoglycan** contained both dibasic amino acids.

L246 ANSWER 75 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS DUPLICATE 3
ACCESSION NUMBER: 1990:434067 BIOSIS
DOCUMENT NUMBER: BR39:81928
TITLE: IN-VITRO SUSCEPTIBILITIES OF BACTEROIDES-GRACILIS

FUSOBACTERIUM-MORTIFERUM AND **FUSOBACTERIUM** -**VARIUM** TO 17 ANTIMICROBIAL AGENTS.
AUTHOR(S): COURCOL R J; LEE K W; DOWNES J; WEXLER H M; BARON E J; FINEGOLD S M
CORPORATE SOURCE: BACTERIOL. LAB., A. CALMETTE HOSP., 59037 LILLE CEDEX, FRANCE.
SOURCE: J. Antimicrob. Chemother., (1990) 26 (1), 157-158. CODEN: JACHDX. ISSN: 0305-7453.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 76 OF 120 LIFESCI COPYRIGHT 2002 CSA
ACCESSION NUMBER: 89:71187 LIFESCI
TITLE: Lectinlike interactions of Fusobacterium nucleatum with human neutrophils.
AUTHOR: Mangan, D.F.; Novak, M.J.; Vora, S.A.; Mourad, J.; Kriger, P.S.
CORPORATE SOURCE: Dep. Dent. Res., Univ. Rochester, Rochester, NY 14642, USA
SOURCE: INFECT. IMMUN., (1989) vol. 57, no. 11, pp. 3601-3611.
DOCUMENT TYPE: Journal
FILE SEGMENT: F; J
LANGUAGE: English
SUMMARY LANGUAGE: English
AB Fusobacterium nucleatum expresses lectinlike adherence factors which mediate binding to a variety of human tissue cells. Adherence is selectively inhibited by galactose, lactose, and N-acetyl-D-galactosamine. In this study, adherence of F. nucleatum to human peripheral blood polymorphonuclear neutrophils (PMNs) was investigated. The results indicated that the fusobacteria adhered to live and metabolically inactivated or fixed PMNs. The results indicate that interaction of F. nucleatum with PMNs is lectinlike and is probably mediated by **fusobacterial** proteins which bind to other human tissue cells. Adherence of F. nucleatum to PMNs in the absence of serum opsonins, such as **antibodies** and complement, may play an important role in PMN recognition and killing of F. nucleatum in the gingival sulcus and in the subsequent release of PMN factors associated with tissue destruction.

L246 ANSWER 77 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:416105 BIOSIS
DOCUMENT NUMBER: BR37:71568
TITLE: SHARING OF **ANTIGENS** BY FUSOBACTERIUM-NUCLEATUM AND FUSOBACTERIUM-NECROPHORUM.
AUTHOR(S): FALKLER W JR; KAUR M; WASFY M; MCMAHON K; MINAH G
CORPORATE SOURCE: UNIV. MD. DENTAL SCH., BALTIMORE, MD.
SOURCE: 67TH GENERAL SESSION OF THE INTERNATIONAL ASSOCIATION FOR DENTAL RESEARCH (IADR), 6TH MEETING OF THE IADR IRISH DIVISION, 72ND ANNUAL MEETING OF THE SCANDINAVIAN ASSOCIATION FOR DENTAL RESEARCH AND THE 26TH ANNUAL MEETING OF THE CONTINENTAL EUROPEAN DIVISION OF THE IADR, DUBLIN, IRELAND, JUNE 28-JULY 1, 1989. J DENT RES, (1989) 68 (SPEC ISSUE JUNE), 964. CODEN: JDREAF. ISSN: 0022-0345.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 78 OF 120 MEDLINE
ACCESSION NUMBER: 89271779 MEDLINE
DOCUMENT NUMBER: 89271779 PubMed ID: 2729931
TITLE: Cell-wall-defective variants of Fusobacterium.

AUTHOR: Johnson C C; Wexler H M; Becker S; Garcia M; Finegold S M
CORPORATE SOURCE: Medical Service, Veterans Administration Wadsworth Medical Center, Los Angeles, California 90073.
SOURCE: ANTIMICROBIAL AGENTS AND CHEMOTHERAPY, (1989 Mar) 33 (3) 369-72.
Journal code: 6HK; 0315061. ISSN: 0066-4804.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198907
ENTRY DATE: Entered STN: 19900309
Last Updated on STN: 19900309
Entered Medline: 19890705

AB The activity of antimicrobial agents against Fusobacterium species has been reported as variable in the literature. For some strains, the inconsistency arises from difficulty in determining the endpoint of growth in agar dilution susceptibility tests. Certain strains persist as a subtle haze beyond the levels of antibiotic that permit conventional colonial growth. We have determined by light and electron microscopy that this haze represents the colonial growth of cell-wall-defective (CWD) variants of the parent Fusobacterium. The CWD forms could be propagated indefinitely in hypertonic medium containing the antibiotic inducing agent. However, when the antibiotic was eliminated, the organisms would revert to their native morphology. Formation of CWD variants was observed in the presence of cell-wall-active drugs (e.g., beta-lactam agents) but not with drugs that work by a different mechanism (e.g., clindamycin or chloramphenicol). Fourteen of 22 F. **varium** strains, 8 of 11 F. mortiferum strains, 2 of 10 F. gonidiaformans strains, and 1 of 4 of F. necrophorum strains could be induced to a CWD form in vitro in the usual agar dilution susceptibility test. Although the clinical significance of CWD variants of Fusobacterium is unknown, they may be a source of confusion in interpreting agar dilution susceptibility tests.

L246 ANSWER 79 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1990:362550 BIOSIS
DOCUMENT NUMBER: BR39:47026
TITLE: EXPERIMENTAL **MODELS** FOR STUDYING THE MICROBIAL ECOLOGY IN THE INTESTINAL TRACT.
AUTHOR(S): RAIBAUD P
CORPORATE SOURCE: LABORATOIRE D'ECOLOGIE MICROBIENNE, CRJ, INRA, 78350 JOUY-EN-JOSAS, FRANCE.
SOURCE: Acta Gastroenterol. Latinoam., (1989) 19 (4), 219-226.
CODEN: AGLTBL. ISSN: 0300-9033.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 80 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1989:374248 BIOSIS
DOCUMENT NUMBER: BR37:53371
TITLE: IN-VITRO SUSCEPTIBILITIES OF BACTEROIDES-GRACILIS **FUSOBACTERIUM**-MORTIFERUM AND **FUSOBACTERIUM** -**VARIUM** TO 18 ANTIMICROBIAL AGENTS.
AUTHOR(S): COURCOL R J; LEE K; DOWNES J; WEXLER H; FINEGOLD S M
CORPORATE SOURCE: WADSWORTH VA MED. CENT., LOS ANGELES, CALIF.
SOURCE: 89TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, NEW ORLEANS, LOUISIANA, USA, MAY 14-18, 1989.
ABSTR ANNU MEET AM SOC MICROBIOL, (1989) 89 (0), 18.
CODEN: ASMACK. ISSN: 0094-8519.
DOCUMENT TYPE: Conference

FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 81 OF 120 MEDLINE
ACCESSION NUMBER: 88296032 MEDLINE
DOCUMENT NUMBER: 88296032 PubMed ID: 3042305
TITLE: Lester R. Dragstedt 1893-1975. Chronic ulcerative colitis. A summary of evidence implicating Bacterium necrophorum as an etiologic agent.
AUTHOR: Anonymous
SOURCE: DISEASES OF THE COLON AND RECTUM, (1988 Aug) 31 (8) 658-64. Journal code: EAB; 0372764. ISSN: 0012-3706.
PUB. COUNTRY: United States
Biography
Article; (CLASSICAL ARTICLE)
Historical
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198809
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19980206
Entered Medline: 19880913

AB Lester Dragstedt was born in Anaconda, Montana, the son of Swedish immigrant parents. His entire college and professional education took place at the University of Chicago, where he received a B.S. degree in 1915, a master's degree in physiology in 1916, a Ph.D. in physiology in 1920, and the M.D. degree (from Rush) in 1921. His first academic appointment was as a physiologist at the State University of Iowa. In 1925 Dragstedt was recruited by Dallas B. Phemister to help design the new University Hospital research facilities on the campus of the University of Chicago. Following completion of this responsibility Phemister appointed Dragstedt as Associate Professor of Surgery, stating, "I can teach surgery to a physiologist; I am interested in teaching physiology to surgeons." In 1947 Dragstedt succeeded Phemister as chairman, a post he occupied until his retirement in 1959. Dragstedt was regarded as a skilled clinician as well as a dexterous and artistic surgeon. But he was particularly recognized for his contributions as physiologist-surgeon in the treatment of diseases of the pancreas, parathyroids, and especially diseases of the stomach. In 1943, he performed a transthoracic vagotomy on a patient with a duodenal ulcer who refused to accept the standard operation, subtotal gastrectomy. A lesser known but classical work of Dragstedt and his coworkers is reproduced here for this series. Dragstedt was the originator of the skin-grafted ileostomy in the treatment of ulcerative colitis. The author describes a complete "take" of the split-thickness graft in four patients, although he observed that the "resulting ileostomy looked somewhat like a penis." One can only surmise about the psychologic disability that would be produced. The stoma could, however, be fitted with an appliance that would minimize the risk of abdominal wall digestion. When reading the article and understanding the experimental studies proposing the possible causative organism of ulcerative colitis, one is impressed by Dragstedt's creative thinking. Dragstedt's renown as a basic scientist was illustrated by his election to the National Academy of Sciences. Following his Chicago retirement he became again a full-time physiologist with appointments as research professor in both the department of surgery and the department of physiology at the University of Florida College of Medicine. Active until the end, he died at his summer home on Elk Lake, Michigan on July 16, 1975.

L246 ANSWER 82 OF 120 MEDLINE

ACCESSION NUMBER: 89046227 MEDLINE
DOCUMENT NUMBER: 89046227 PubMed ID: 3188735
TITLE: [Influence of the oral administration of indigenous microorganisms on the resistance of mice to Salmonella infection].
Vliianie peroral'nogo vvedeniia indigennykh mikroorganizmov na ustoichivost' myshei k sal'monelleznoi infektsii.
AUTHOR: Shkarupeta M M; Korshunov V M; Savenkova V T; Pinegin B V
SOURCE: ZHURNAL MIKROBIOLOGII, EPIDEMIOLOGII I IMMUNOBIOLOGII, (1988 Jul) (7) 46-50.
Journal code: Y9O; 0415217. ISSN: 0372-9311.
PUB. COUNTRY: USSR
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: Russian
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198811
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19881130

AB The influence of the oral administration of killed bifidobacteria, lactobacteria, bacteroids and fusobacteria on the anti- Salmonella resistance of mice, infected orally with S. dublin, was studied. Bifidobacteria and lactobacteria were shown to produce a dose-dependent immunostimulating effect. The oral administration of killed bifidobacteria and lactobacteria led to the enhanced resistance of mice to Salmonella infection. The oral administration of killed bifidobacteria was conductive to the normalization of the intestinal microflora in dysbacteriosis developing in cases of Salmonella infection. Bacteroids and fusobacteria were found to possess no such effect.

L246 ANSWER 83 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1988:335047 BIOSIS
DOCUMENT NUMBER: BA86:41598
TITLE: STUDIES ON **ENDOTOXIN** EXTRACTED FROM **FUSOBACTERIUM** BY THE EDTA AND TCA METHODS.
AUTHOR(S): KAKU N
CORPORATE SOURCE: DEP. BACTERIOL., OSAKA DENT. UNIV., 1-47 KYOBASHI, HIGASHI-KU, OSAKA 540, JPN.
SOURCE: J OSAKA ODONTOL SOC, (1988) 51 (1), 1-12.
CODEN: SIGAAE. ISSN: 0030-6150.
FILE SEGMENT: BA; OLD
LANGUAGE: Japanese

AB Endoxins were isolated from the ATCC strain of Fusobacterium nuclatum, Fusobacterium necrophorum and **Fusobacterium varium** by the EDTA and TCA methods and the properties of each **endotoxin** obtained were compared. The **endotoxins** extracted by EDTA were lower in protein content than those extracted by TCA. Among the two preparations extracted by the different methods from the same strain, the antigenicity was examined using the precipitation test and serological homogenity was partially observed in both. All of the **endotoxins** showed different patterns with SDS polyacrilamide gel electrophoresis analysis and showed several major bands before and after 50 kilodaltons. Endotoxic activity, as measured by the Shwartzman reaction, was not present in either preparation obtained from Fusobacterium nucleatum. Acute toxicity was present in all preparations. Also, limulus lysate activity was present in all samples, but the activity of **endotoxins** isolated by the EDTA method was lower than that of **endotoxins** isolated by the TCA method.

L246 ANSWER 84 OF 120 CABA COPYRIGHT 2002 CABI

ACCESSION NUMBER: 87:52045 CABA
DOCUMENT NUMBER: 871330588
TITLE: Intergeneric protoplast fusion between **Fusobacterium varium** and Enterococcus faecium for enhancing dehydrodivanillin degradation
AUTHOR: Chen, W.; Ohmiya, K.; Shimizu, S.
CORPORATE SOURCE: Dep. Food Sci. Technol., Sch. Agric., Nagoya Univ., Chikusa, Nagoya, 464, Japan.
SOURCE: Applied and Environmental Microbiology, (1987) Vol. 53, No. 3, pp. 542-548. 30 ref.
ISSN: 0099-2240
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Intergeneric protoplast fusion between **F. varium** (Pcs Glu+) and E. faecium (Pcr Glu-) was performed under strictly anaerobic conditions to improve dehydrodivanillin (DDV) degradation. The fusion frequency obtained from the selective medium (Pc+ Glu-) was c. 0.9 x 10-5 to 1.3 x 10-5. The 7 fusants isolated were all Gram-negative anaerobes with rod shapes like that of **F. varium** and with main phenotypical properties of cocci like those of E. faecium such as esculin and starch hydrolysis, milk clotting, and lactate production. Five fusants showed enhanced DDV degradation activities that were 2-4 times higher than those of parental strains. Genetic relatedness between a fusant (FE7) and the parents was estimated by DNA-DNA Southern blot hybridization with 32P-labelled chromosomal DNA fragments of **F. varium** and E. faecium as respective probes. The fusant FE7 presented a very high cross-hybridization with both probes, indicating a high DNA homology between the fusant and both parental strains. Almost all the fusants obtained here have stably kept the properties described above for about 2 years. These results suggest that intergeneric gene transfer takes place through protoplast fusion and that the fusants that were obtained are stable recombinants.

L246 ANSWER 85 OF 120 MEDLINE
ACCESSION NUMBER: 88133318 MEDLINE
DOCUMENT NUMBER: 88133318 PubMed ID: 3435242
TITLE: Comparative immunological studies on Corynebacterium parvum polysaccharide and Fusobacterium **varium** lipopolysaccharide.
AUTHOR: Marx A; Salageanu A; Olinescu A; Gancevici G
SOURCE: ARCHIVES ROUMAINES DE PATHOLOGIE EXPERIMENTALES ET DE MICROBIOLOGIE, (1987 Jan-Mar) 46 (1) 57-65.
Journal code: 8EM; 0421056. ISSN: 0004-0037.
PUB. COUNTRY: Romania
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198802
ENTRY DATE: Entered STN: 19900308
Last Updated on STN: 19900308
Entered Medline: 19880229

L246 ANSWER 86 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 87230489 EMBASE
DOCUMENT NUMBER: 1987230489
TITLE: Synergistic aerobic and anaerobic infections.
AUTHOR: Brook I.
CORPORATE SOURCE: Department of Pediatrics, Uniformed Services, University of the Health Sciences, Bethesda, MD, United States

SOURCE: Clinical Therapeutics, (1987) 10/SUPPL. A (19-35).
ISSN: 0149-2918 CODEN: CLTHDG
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
037 Drug Literature Index
LANGUAGE: English

AB Encapsulation affects the virulence and survival of anaerobic bacteria and their protection from phagocytosis. More encapsulated Bacteroides strains and anaerobic and facultative gram-positive cocci are isolated from patients with clinical infections than from healthy people. The pathogenicity of Bacteroides, Fusobacterium, Clostridium, and cocci isolates was demonstrated by their ability to induce subcutaneous abscesses in mice. Encapsulated Bacteroides, Fusobacterium, and cocci isolates generally induced abscesses, whereas nonencapsulated organisms did not. When strains that had fewer than 1% encapsulated organisms were inoculated with other viable or nonviable encapuslated bacteria, many survived in the abscesses and became heavily encapsulated. These strains were then able to induce abscesses when injected alone. Encapuslated Bacteroides species and anaerobic cocci induced bacteremia and translocation and increased the mortality in infected animals more often than did nonencapsulated forms of the same strain. In studies of selective antimicrobial therapy and quantitative cultures of abscesses, it was determined that possession of a capsule generally made Bacteroides species more important in mixed infections than their aerobic counterparts. In vivo synergy was seen between encapsulated Bacteroides species and all tested aerobic bacteria and most anaerobic and facultative gram-positive cocci as well as between most of these cocci and Pseudomonas aeruginosa or Staphylococcus aureus. It is concluded that encapsulated anaerobic bacteria have an important pathogenic role in polymicrobial infections.

L246 ANSWER 87 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 87230488 EMBASE
DOCUMENT NUMBER: 1987230488
TITLE: In vitro activity of selected antibiotics against anaerobes.
AUTHOR: Wexler H.M.; Finegold S.M.
CORPORATE SOURCE: Veterans Administration Medical Center, UCLA School of Medicine, Los Angeles, CA, United States
SOURCE: Clinical Therapeutics, (1987) 10/SUPPL. A (12-18).
ISSN: 0149-2918 CODEN: CLTHDG
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 004 Microbiology
037 Drug Literature Index
LANGUAGE: English

AB Testing antibiotics for their activity against microorganisms is fraught with problems. The various methods and media yield different results, and controversy exists as to which is the most reliable. The technique used in our laboratory has shown wide differences in the susceptibility patterns of Bacteroides strains and other anaerobes to different antibiotics. Particularly with respect to the third-generation cephalosporins, reliable clinical data are needed to determine which in vitro tests most accurately predict clinical efficacy.

L246 ANSWER 88 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 87044768 EMBASE
DOCUMENT NUMBER: 1987044768
TITLE: Metabolic induction of experimental **ulcerative colitis** by inhibition of fatty acid oxidation.

AUTHOR: Roediger W.E.W.; Nance S.
CORPORATE SOURCE: Department of Surgery, The Queen Elizabeth Hospital, Woodville, S.A. 5011, Australia
SOURCE: British Journal of Experimental Pathology, (1986) 67/6 (773-782).
CODEN: BJEPA5
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
005 General Pathology and Pathological Anatomy
048 Gastroenterology
029 Clinical Biochemistry
LANGUAGE: English

AB There is some evidence that failure of fatty acid or .beta.-oxidation in the epithelium of the colonic mucosa is associated with the development of **ulcerative colitis**. We tested the hypothesis that inhibition of fatty acid oxidation in the colonic mucosa of the rat reproduces the histological, clinical and biochemical lesions of acute **ulcerative colitis** of man. A specific inhibitor of .beta.-oxidation, sodium 2-bromo-octanoate, was instilled rectally for 5 days or exposed to isolated colonic epithelial cells which were subsequently tested for their ability to .beta.-oxidize n-**butyrate**. Weight loss, bloody diarrhoea and histological lesions occurred with 2-bromo-octanoate treated rats but not control animals. Ketogenesis and 14CO2 production was inhibited by 2-bromo-octanoate. Of 12 animals mucosal ulceration developed in six out of eight surviving animals and in all four animals that died. Ulceration, mucus cell depletion, vessel dilatation and increases of inflammatory cells were the most prominent histological changes. Present observations indicate that inhibition of .beta.-oxidation produces acute colitis and suggests that inhibition of .beta.-oxidation is primary rather than secondary in the genesis of **ulcerative colitis**. A search for agents producing such biochemical lesions in man should be undertaken.

L246 ANSWER 89 OF 120 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 87:2901 CABA
DOCUMENT NUMBER: 871321037
TITLE: Protoplast formation and regeneration of dehydrodivanillin-degrading strains of **Fusobacterium varium** and Enterococcus faecium
AUTHOR: Chen, W.; Ohmiya, K.; Shimizu, S.
CORPORATE SOURCE: Dep. Food Sci. Technol., Sch. Agric., Nagoya Univ., Chikusa, Nagoya 464, Japan.
SOURCE: Applied and Environmental Microbiology, (1986) Vol. 52, No. 4, pp. 612-616. 27 ref.
ISSN: 0099-2240
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Two strs. of rumen anaerobes isolated from dehydrovanillin-degrading cultures were identified as **F. varium** and E. faecium. These organisms degraded dehydrovanillin synergistically to 5-carboxymethylvanillin and vanillic acid. Specific conditions for protoplast formation and cell wall regeneration for both bacteria were determined, under strictly anaerobic conditions, to be as follows: (i) The cell wall of each bacterium in yeast extract medium was loosened by adding penicillin G during early log-phase growth; (ii) The cell wall of **F. varium** was lysed by lysozyme (1 mg/ml) in glycerol (0.2 M)-phosphate buffer (0.05 M, pH 7.0). The addition of NaCl (0.08 M) with lysozyme was necessary for lysis of E. faecium in this solution.

Almost all cells were converted to protoplasts after 2 h of incubation at 37 deg C; (iii) Regeneration of both protoplasts was 20-30% on an agar-containing yeast extract medium.

L246 ANSWER 90 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1986:125908 CAPLUS
DOCUMENT NUMBER: 104:125908
TITLE: Quantitative gas chromatographic analysis of volatile fatty acids in spent culture media and body fluids
AUTHOR(S): Van den Bogaard, Anthony E.; Hazen, Mathew J.; Van Boven, Cees P.
CORPORATE SOURCE: Dep. Med. Microbiol., Univ. Limburg, Maastricht, 6200 MD, Neth.
SOURCE: J. Clin. Microbiol. (1986), 23(3), 523-30
CODEN: JCMIDW; ISSN: 0095-1137
DOCUMENT TYPE: Journal
LANGUAGE: English
AB Three stationary phases were compared for volatile fatty acid anal. of aq. solns. and 4 methods of pretreating samples were compared for gas chromatog. Quant. anal. could be done accurately by using Carbowax as the stationary phase after pretreatment of spent culture media with Dowex columns. If only qual. anal. is required (e.g., for presumptive diagnosis of anaerobic infections), ether extn. and headspace anal. are equally suitable. The overall variation coeff. for volatile fatty acid prodn. by 4 ref. strains of obligately anaerobic bacteria after 24 h of incubation was approx. 10%.

L246 ANSWER 91 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1986:240267 BIOSIS
DOCUMENT NUMBER: BA82:4771
TITLE: EXPERIMENTAL ANIMAL **MODEL** OF **ULCERATIVE COLITIS** USING ESCHERICHIA-COLI.
AUTHOR(S): HOTTA T
CORPORATE SOURCE: KAWARAMACHI-HIROCOJI, KAMIGYOKU, KYOTO 602, JAPAN.
SOURCE: J KYOTO PREFECT UNIV MED, (1986) 95 (3), 325-338.
CODEN: KFIZAO.
FILE SEGMENT: BA; OLD
LANGUAGE: English
AB Experimental animal models of human ulcerative colitis were prepared using E. coli **lipopolysaccharide** (LPS) or boiled E. coli derived from the rabbit's own feces, and the course was serially investigated by colonofiberscopic examinations and biopsies. Rabbits were sensitized by LPS and challenged with intrarectal instillation of LPS after administering 1% formalin enema. Petechiae appeared after 8 h, and ulcers and bleeding on the 3rd day. Mild macroscopic changes continued for about 2 weeks. By repeating the LPS enema after the initial treatment, the colitis was maintained for over 1 month. Sensitized control rabbits given LPS enema without formalin treatment had no macroscopic changes, and non-sensitized rabbits with formalin and LPS enema only showed mild transient changes. E. coli-sensitized rabbits showed long-standing colitis induced only by formalin enema. The **endotoxin** level in the blood during the experiment was higher in the non-sensitized control rabbits than in the LPS-sensitized group. Fibrinogen and PTT levels were increased at 24 and 72 h, particularly in the control rabbits. Tissue fibrinolysis of the colon increased significantly with the development of mucosal damage. Continuous stimulation of colonic mucosa by bacterial antigen in the rabbits sensitized with intestinal bacteria or its components was considered to be an important factor in chronic ulcerative colitis.

L246 ANSWER 92 OF 120 MEDLINE
ACCESSION NUMBER: 87044074 MEDLINE
DOCUMENT NUMBER: 87044074 PubMed ID: 3776094
TITLE: Generation of immunity against **Fusobacterium** necrophorum in mice inoculated with extracts containing leucocidin.
AUTHOR: Emery D L; Vaughan J A
SOURCE: VETERINARY MICROBIOLOGY, (1986 Sep) 12 (3) 255-68. Journal code: XBW; 7705469. ISSN: 0378-1135.
PUB. COUNTRY: Netherlands
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198612
ENTRY DATE: Entered STN: 19900302
Last Updated on STN: 19900302
Entered Medline: 19861202

AB The capacity of extracts from toxigenic and non-toxigenic ruminant strains of Fusobacterium necrophorum to protect against challenge with homologous and heterologous bacteria was examined in mice. The numbers of F. necrophorum which were infective or lethal for mice increased 5- to 8-fold in animals which had been previously inoculated with complete Freund's adjuvant (FCA). Although preparations containing lipopolysaccharide (LPS) and outer membrane proteins (OMP) from several strains gave protection against a non-toxigenic strain (FnB-3), they did not significantly immunize mice against a challenge infection with a toxigenic bovine strain, FnB-1. Only material which had been prepared by gel filtration of 18-h liquid culture supernates of toxigenic F. necrophorum elicited significant immunity against homologous challenge with FnB-1. This preparation contained LPS and the majority of the leucotoxic activity. However, passive protection was not afforded to mice inoculated with bovine or rabbit sera which possessed high neutralization titres against the leucocidin.

L246 ANSWER 93 OF 120 EMBASE
ACCESSION NUMBER: 86162560 EMBASE
DOCUMENT NUMBER: 1986162560
TITLE: Effect of 5-aminosalicylic acid (5-ASA) and other salicylates on short-chain fat metabolism in the colonic mucosa. Pharmacological implications for **ulcerative colitis**.
AUTHOR: Roediger W.; Schapel G.; Lawson M.; et al.
CORPORATE SOURCE: Cell Physiology Laboratory, Queen Elizabeth Hospital and University of Adelaide, Woodville, S.A. 5011, Australia
SOURCE: Biochemical Pharmacology, (1986) 35/2 (221-225). CODEN: BCPCA6
COUNTRY: United Kingdom
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
048 Gastroenterology
029 Clinical Biochemistry
LANGUAGE: English

AB 5-Aminosalicylic acid (5-ASA) suppressed nitrite-stimulated oxidation of the fatty acid n-**butyrate** in a dose-dependent manner in isolated human and rat colonic epithelial cells. 4-ASA had one-sixth of the capacity of 5-ASA and sulphapyridine (SP) little of the capacity of 5-ASA to suppress fatty acid oxidation in human colonic epithelial cells. Sulphasalazine (SASP), azodisalicylic acid (ADS), acetyl-5-ASA and acetyl salicylic acid (ASA) did not suppress fatty acid oxidation in rat

colonocytes. The suppression index of fatty acid oxidation (SIFO) of respective salicylic acids correlated with the reported clinical effectiveness of each drug against **ulcerative colitis** (UC). The capacity of 5-ASA to affect nitrate-stimulated oxidation of fat in the colonic mucosa suggests that nitrite ions and control of fatty acid oxidation play a central role in the development and therapy of active UC.

L246 ANSWER 94 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1986:259781 BIOSIS
DOCUMENT NUMBER: BA82:14530
TITLE: THE RELATIONSHIP BETWEEN **FUSOBACTERIUM** SPECIES AND OTHER FLORA IN MIXED **INFECTION**.
AUTHOR(S): BROOK I; WALKER R I
CORPORATE SOURCE: NAVAL MED. RES. INST., BETHESDA, MD 20814, USA.
SOURCE: J MED MICROBIOL, (1986) 21 (2), 93-100.
CODEN: JMMIAV. ISSN: 0022-2615.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB Mixed **infections** with three Fusobacterium species and seven other bacterial species were studied in a subcutaneous abscess **model** in mice. Fifteen Fusobacterium isolates (eight F. nucleatum, four F. necrophorum, and three **F. varium**) and one isolate each of Bacteroides fragilis, B. asaccharolyticus, Staphylococcus aureus, Group A .beta.-haemolytic streptococcus, Escherichia coli, Klebsiella pneumoniae and Pseudomonas aeruginosa were studied. Electron micrographs showed the presence of a thin mucopolysaccharide wall before and after inoculation into mice in 12 isolates which included all of 11 Fusobacterium isolates that induced subcutaneous abscesses. After co-inoculation of Fusobacterium isolates with other species and selective therapy with antimicrobial agents, S. aureus and K. pneumoniae were found to be of equal or greater importance in abscess induction than were Fusobacterium isolates, while Fusobacterium isolates were found to be more important than Group A streptococci and E. coli. Mutual enhancement of the numbers of organisms in mixed **infections** was observed with Fusobacterium spp. and K. pneumoniae, P. aeruginosa or Bacteriodes spp. Suppression of Fusobacterium spp. was noticed only when they were co-inoculated with Group A streptococci. The additive or synergistic capabilities of Fusobacterium species highlighted their potential pathogenicity in **infection**.

L246 ANSWER 95 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1985:403243 CAPLUS
DOCUMENT NUMBER: 103:3243
TITLE: Compound and composition for therapeutic or diagnostic use
INVENTOR(S): Karlsson, Karl Anders; Lindberg, Alf Anton
PATENT ASSIGNEE(S): Swed.
SOURCE: Eur. Pat. Appl., 10 pp.
CODEN: EPXXDW
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
EP 133170	A2	19850213	EP 1984-850211	19840704
EP 133170	A3	19890201		
R: AT, BE, CH, DE, FR, GB, IT, LI, NL, SE				
AU 8430493	A1	19850117	AU 1984-30493	19840711

AU 581850	B2	19890309		
FI 8402814	A	19850116	FI 1984-2814	19840712
DK 8403473	A	19850116	DK 1984-3473	19840713
JP 60064993	A2	19850413	JP 1984-144520	19840713

PRIORITY APPLN. INFO.:. SE 1983-4006 19830715

AB A compd., compn., and method are described for the diagnosis, prophylaxis, and treatment of bacterial infections in mammals, for use as disinfectant, and for the purifn. of bacteria acceptor structures. For therapeutic treatment, the compd. (in combination with a pharmaceutically acceptable carrier) is administered to the mammal. For detg. the presence of bacteria in the gastrointestinal tract of mammals, the degree of interaction between the bacteria and the compd. is detd. Thus, to TLC plates covered with treated silica gel mixed with bacterial receptors (purified or in crude exts.) are transferred 125I-labeled bacteria (Propionibacterium granulosum or P. freudenreichii). Following TLC and sepn. of glycolipids, autoradiog. was carried out for the detection of glycolipids bound to the receptors. The receptors used were glycolipids of human erythrocytes, human meconium, monkey intestine, dog small intestine, and rabbit and guinea pig small intestine.

L246 ANSWER 96 OF 120 MEDLINE
ACCESSION NUMBER: 85120236 MEDLINE
DOCUMENT NUMBER: 85120236 PubMed ID: 3970414
TITLE: Development of enzyme-linked immunosorbent assays for the detection of **Fusobacterium** necrophorum antibody in animal sera.
AUTHOR: Evans J W; Berg J N
SOURCE: AMERICAN JOURNAL OF VETERINARY RESEARCH, (1985 Jan) 46 (1) 132-5.
Journal code: 40C; 0375011. ISSN: 0002-9645.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198503
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850307

AB Enzyme-linked immunosorbent assays (ELISA) for the detection of Fusobacterium necrophorum antibody in the sera of rabbits, cattle, and sheep were developed, using a ribosome-rich extract (RRE) from F necrophorum as the antigen. Test character, including optimal antigen dilution and substrate incubation periods, was established, using rabbit, bovine, and ovine antisera produced against RRE from isolates of F necrophorum. Rabbit antisera produced against 7 other species of bacteria were used to test the specificity of the F necrophorum RRE antigen. Cross-reactivity was not detected. Sera from 50 feedlot cattle were examined with the bovine ELISA. Of the 50 samples, 43 (88%) were positive for F necrophorum antibody. The ELISA developed in this study were sensitive and specific and appear to be readily adaptable to serologic investigations of F necrophorum.

L246 ANSWER 97 OF 120 MEDLINE
ACCESSION NUMBER: 85120234 MEDLINE
DOCUMENT NUMBER: 85120234 PubMed ID: 3918487
TITLE: Identification of common antigens in ribosome-rich extracts from **Fusobacterium** necrophorum.
AUTHOR: Berg J N; Evans J W
SOURCE: AMERICAN JOURNAL OF VETERINARY RESEARCH, (1985 Jan) 46 (1) 127-31.

Journal code: 40C; 0375011. ISSN: 0002-9645.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198503
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850307

AB Ribosome-rich extracts (RRE) were prepared by differential and ultracentrifugation from 25 bovine and 6 ovine isolates of Fusobacterium necrophorum (FN) including both biotypes A and B. A pooled rabbit antiserum was prepared against whole-cell and sonicated whole-cell bacterins of F necrophorum isolate FN 3080, and a 2nd pooled rabbit antiserum was prepared against a RRE of FN 3080. The RRE of the 25 bovine isolates were tested against the FN 3080 whole-cell antiserum, using Ouchterlony double-immunodiffusion procedures. One to 3 precipitin lines were observed with the 25 isolates. The individual bovine isolates were found to have lines of identity with 5 to 21 of the other 24 isolates. The 25 bovine isolates and the 6 ovine isolates were then compared, using the FN 3080 RRE antiserum. One to 3 precipitin lines were observed for the 31 isolates with the RRE antiserum, and lines of identity were observed between all 31 of the isolates. These results indicated that common antigens are present in the RRE from a wide variety of F necrophorum isolates including both A and B biotypes.

L246 ANSWER 98 OF 120 MEDLINE
ACCESSION NUMBER: 85262781 MEDLINE
DOCUMENT NUMBER: 85262781 PubMed ID: 3894509
TITLE: The weak immunogenicity of **Fusobacterium** necrophorum.
AUTHOR: Smith G R; Turner A; Murray L G; Oliphant J C
SOURCE: JOURNAL OF HYGIENE, (1985 Aug) 95 (1) 59-68.
Journal code: IEF; 0375374. ISSN: 0022-1724.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 198509
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19900320
Entered Medline: 19850911

AB Three of four extreme methods of immunization completely failed to protect mice against challenge with the homologous strain of Fusobacterium necrophorum. Unsuccessful vaccines included (1) broth culture killed by mild heat and emulsified with Freund's complete adjuvant, and (2) a homogenate of heavily infected mouse brains, inactivated by mild heat and given in two doses. Also unsuccessful as a method of immunization was the production of a severe subcutaneous infection with F. necrophorum, followed by curative treatment with metronidazole. Slight but significant protection against subcutaneous challenge resulted, however, from two such infections given in rapid succession. It would appear that the main virulence factors of F. necrophorum are only weakly immunogenic, and the experiments give little encouragement to the prospect of an effective necrobacillosis vaccine.

L246 ANSWER 99 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1985:142711 BIOSIS
DOCUMENT NUMBER: BR29:32707
TITLE: PATHOGENICITY OF FUSOBACTERIUM-SPP.

AUTHOR(S): BROOK I; WALKER R I
CORPORATE SOURCE: NAV. MED. RES. INST., BETHESDA, MD.
SOURCE: 85TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, LAS VEGAS, NEV., USA, MAR. 3-7, 1985. ABSTR ANNU MEET AM SOC MICROBIOL, (1985) 85 (0), 56. CODEN: ASMACK. ISSN: 0094-8519.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 100 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1985:142432 BIOSIS
DOCUMENT NUMBER: BR29:32428
TITLE: BETA LACTAMASE PRODUCTION BY CLINICAL ISOLATES OF **FUSOBACTERIUM**.
AUTHOR(S): RANKIN D R; LEE D T; ROSENBLATT J E
CORPORATE SOURCE: MAYO CLINIC, ROCHESTER, MINN. 55905.
SOURCE: 85TH ANNUAL MEETING OF THE AMERICAN SOCIETY FOR MICROBIOLOGY, LAS VEGAS, NEV., USA, MAR. 3-7, 1985. ABSTR ANNU MEET AM SOC MICROBIOL, (1985) 85 (0), 9. CODEN: ASMACK. ISSN: 0094-8519.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 101 OF 120 MEDLINE
ACCESSION NUMBER: 84213281 MEDLINE
DOCUMENT NUMBER: 84213281 PubMed ID: 6327778
TITLE: Colonic bicarbonate output as a test of disease activity in ulcerative **colitis**.
AUTHOR: Roediger W E; Lawson M J; Kwok V; Grant A K; Pannall P R
SOURCE: JOURNAL OF CLINICAL PATHOLOGY, (1984 Jun) 37 (6) 704-7. Journal code: HT3; 0376601. ISSN: 0021-9746.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 198407
ENTRY DATE: Entered STN: 19900320
Last Updated on STN: 19990129
Entered Medline: 19840713

AB No available test objectively measures impairment of function of the inflamed colonic mucosa in ulcerative colitis. To study function we assessed rectal bicarbonate output by rectal dialysis in the presence of water and bacterial fatty acid (n-butyrate) in 21 controls, 18 patients with acute ulcerative colitis, 12 patients with ulcerative colitis in remission, and 12 patients with other forms of colitis. In acute ulcerative colitis, compared with controls, bicarbonate output and pH was reduced (p less than 0.001); stimulated bicarbonate output with bacterial fatty acid (incremental bicarbonate output) was reduced by 80% in acute ulcerative colitis (p less than 0.01). Results indicate that bicarbonate output is a useful and selective test of mucosal function in acute ulcerative colitis. A diminished incremental bicarbonate output with n-butyrate supports the view of inadequate oxidation of bacterial fatty acids in vivo by the mucosa in ulcerative colitis. Whether the test will prove to be an index of prognosis or will aid choice between medical or surgical therapy requires further study.

L246 ANSWER 102 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1984:451508 CAPLUS

DOCUMENT NUMBER: 101:51508
TITLE: Pleomorphism of fusobacteria isolated from the cockroach hindgut
AUTHOR(S): Foglesong, M. A.; Cruden, D. L.; Markovetz, A. J.
CORPORATE SOURCE: Dep. Microbiol., Univ. Iowa, Iowa City, IA, 52242, USA
SOURCE: J. Bacteriol. (1984), 158(2), 474-80
CODEN: JOBAAY; ISSN: 0021-9193
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Fusobacteria are commonly isolated from the hindgut of the cockroach Eublaberus posticus. Eleven strains isolated from E. posticus were keyed to 4 species, Fusobacterium necrophorum, F. varium, F. gonidiaformans, and F. prausnitzii using current taxonomic criteria. With the exception of F. gonidiaformis, all species showed rods with swollen centers and large bodies. The pleomorphism of F. varium was examd. by phase microscopy and scanning and transmission electron microscopy. The pleomorphic process begins with a gradual swelling at the center of the rod until a large round body is formed. Some of these round bodies then fragment, giving rise to rod-shaped cells. When 10% yeast ext. was added to growth media, pleomorphism was not induced. A dialyzable factor was found to account for this observation. Fermn. of [1-14C]glutamic acid gives rise to **butyrate** labeled in the carboxyl carbon, indicating that **butyrate** is formed by the hydroxyglutarate pathway which may be characteristic for the genus Fusobacterium.

L246 ANSWER 103 OF 120 CABA COPYRIGHT 2002 CABI DUPLICATE 4
ACCESSION NUMBER: 84:74056 CABA
DOCUMENT NUMBER: 842241379
TITLE: The pathogenic properties of **Fusobacterium** and Bacteroides species from wallabies and other sources
AUTHOR: Smith, G. R.; Oliphant, J. C.; Parsons, R.
CORPORATE SOURCE: Nuffield Lab. Comp. Med., Inst. Zool., Regent's Park, London NW1 4RY, UK.
SOURCE: Journal of Hygiene, (1984) Vol. 92, No. 2, pp. 165-175. 38 ref.
DOCUMENT TYPE: Journal
LANGUAGE: English

AB Intracerebral inoculation was more effective than i/p, i/v or s/c inoculation for producing lethal infections with F. necrophorum in mice. Strains varied in virulence but, of 5 examined, two had LD50 values as low as 8000 and 14 000 viable organisms. Profuse bacterial multiplication in the brain was demonstrated. I/v vaccination with a single large dose of heat-killed whole culture or washed bacterial cells failed to protect against intracerebral challenge. Intracerebral injection of other fusobacteria (F. nucleatum, **F. varium** and F. necrogenes) and of 22 strains belonging to 10 Bacteroides spp. was without apparent effect on mice, except for a slight transient illness in some animals given B. fragilis. This organism (5 strains) differed from the other Bacteroides spp. tested, which included 8 strains belonging to the fragilis group, in being eliminated more slowly from the mouse brain-a point that may be relevant to the special pathogenicity of B. fragilis in endogenous infections in man. There was no evidence that B. fragilis multiplied in the brain or that i/v vaccination with a large dose of heat-killed homologous culture affected the rate at which it was eliminated.

L246 ANSWER 104 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 83248977 EMBASE
DOCUMENT NUMBER: 1983248977

TITLE: [Antibiotic susceptibility testing of anaerobes].
ETUDE DE LA SENSIBILITE ANTIBIOTIQUE DES ANAEROBES STRICTS.
AUTHOR: Dubreuil L.; Devos J.; Neut Ch.; Romond Ch.
CORPORATE SOURCE: UER Pharm., 59045 Lille, France
SOURCE: Medecine et Maladies Infectieuses, (1983) 13/8 (478-483).
CODEN: MMAIB5
COUNTRY: France
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
004 Microbiology
LANGUAGE: French
SUMMARY LANGUAGE: English
AB Ninety clinical isolates of anaerobic bacteria were tested for susceptibility by a twofold dilution method in brain-heart broth. Penicillin G remained the most active antibacterial agent against gram-positive strains, like metronidazole for Bacteroides fragilis. Cefoxitin appeared to be active on all strains tested except for one Fusobacterium varium and all Clostridium difficile. Resistance to clindamycin was found for few strains in Bacteroides fragilis group and Clostridium difficile. Chloramphenicol proved to be the most effective agent in vitro with only two resistant strains (Cl. difficile, F. varium).

L246 ANSWER 105 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS DUPLICATE 5
ACCESSION NUMBER: 1982:32100 BIOSIS
DOCUMENT NUMBER: BR22:32100
TITLE: GRAM NEGATIVE ANAEROBIC BACILLI THEIR ROLE IN **INFECTION** AND PATTERNS OF SUSCEPTIBILITY TO ANTI MICROBIAL AGENTS 2. LITTLE KNOWN **FUSOBACTERIUM** SPECIES AND MISCELLANEOUS GENERA.
AUTHOR(S): GEORGE W L; KIRBY B D; SUTTER V L; CITRON D M; FINEGOLD S M
CORPORATE SOURCE: BUILDING 500, ROOM 3658, VETERANS ADM. WADSWORTH MED. CENT., LOS ANGELES, CALIF. 90073.
SOURCE: Rev. Infect. Dis., (1981) 3 (3), 599-626.
CODEN: RINDDG. ISSN: 0162-0886.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 106 OF 120 CAPLUS COPYRIGHT 2002 ACS
ACCESSION NUMBER: 1983:591177 CAPLUS
DOCUMENT NUMBER: 99:191177
TITLE: Identification and classification of anaerobic bacteria by gas-liquid chromatography
AUTHOR(S): Anon.
CORPORATE SOURCE: Tunisia
SOURCE: Rev. Fac. Sci. Tunis (1981), 1, 188-96
CODEN: RFSTDY
DOCUMENT TYPE: Journal
LANGUAGE: Arabic
AB A method has been developed for identification of anaerobic bacteria on the basis of gas-liq. chromatog. anal. of the volatile fatty acids produced by different species as end products of carbohydrate fermn. About 20 strains of anaerobes of the genera Clostridium, Ristella, Bifidobacterium, Sphaerophorus, Staphylococcus, and Streptococcus were examd. and gave species-specific patterns of volatile fatty acid compns. Seven major fatty acids were detected: acetic, propionic, isobutyric, **butyric**, valeric, isovaleric, and isocaproic acids. The method may be used as a routine test for the diagnostic identification of anaerobic bacteria.

L246 ANSWER 107 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS

ACCESSION NUMBER: 1980:233486 BIOSIS
DOCUMENT NUMBER: BA70:25982
TITLE: FATTY-ACIDS AND NEUTRAL SUGARS PRESENT IN LIPO POLY SACCHARIDES ISOLATED FROM **FUSOBACTERIUM**-SPP.
AUTHOR(S): HOFSTAD T; SKAUG N
CORPORATE SOURCE: MIKROBIOL. AVD., MFH-BYGGET, N-5016 HAUKELAND SYKEHUS, NORW.
SOURCE: ACTA PATHOL MICROBIOL SCAND SECT B MICROBIOL, (1980) 88 (2), 115-120.
CODEN: APBMDF. ISSN: 0304-131X.
FILE SEGMENT: BA; OLD
LANGUAGE: English

AB **Lipopolysaccharides** (LPS) extracted from cells of F. necrophorum, F. mortiferum, F. gonidiaformans, **F. varium** and single strains of F. naviforme and F. russii were analyzed for sugars and fatty acids. All preparations contained a monosaccharide tentatively identified as L-glycero-D-mannoheptose, but showed variation with respect to the presence of glucose, galactose, rhamnose and another heptose isomer, tentatively identified as L-glycero-D-mannoheptose. L-glycero-D-mannoheptose was a monosaccharide of great abundance in all LPS examined. 2-Keto-3-deoxy-octonate and glucosamine were present in all LPS preparations. The fatty acids present were 3-hydroxy-tetradecanoic acid as the main component, and n-tetradecanoic acid. Some LPS contained n-hexadecanoic acid. All the strains of F. nucleatum contained 3-hydroxy-hexadecanoic acid as a group-specific LPS constituent.

L246 ANSWER 108 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1980:4284 BIOSIS
DOCUMENT NUMBER: BR18:4284
TITLE: SEROLOGICAL RESPONSES TO **ANTIGENS** OF BACTEROIDACEAE.
AUTHOR(S): HOFSTAD T
CORPORATE SOURCE: DEP. MICROBIOL., GADE INST., UNIV. BERGEN., BERGEN, NORW.
SOURCE: Microbiol. Rev., (1979) 43 (1), 103-115.
CODEN: MBRED3. ISSN: 0146-0749.
FILE SEGMENT: BR; OLD
LANGUAGE: English

L246 ANSWER 109 OF 120 MEDLINE DUPLICATE 6
ACCESSION NUMBER: 79225430 MEDLINE
DOCUMENT NUMBER: 79225430 PubMed ID: 223235
TITLE: Investigation of the immune response to aerobic and anaerobic intestinal bacteria in a patient with Crohn's disease.
AUTHOR: Danielsson D; Kjellander J; Persson S; Wallensten S
SOURCE: SCANDINAVIAN JOURNAL OF INFECTIOUS DISEASES. SUPPLEMENTUM, (1979) (19) 52-60.
Journal code: UCY; 0251025. ISSN: 0300-8878.
PUB. COUNTRY: Sweden
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197909
ENTRY DATE: Entered STN: 19900315
Last Updated on STN: 19980206
Entered Medline: 19790927

AB The immune response to aerobic and anaerobic intestinal bacteria in a patient with Crohn's disease with an intestinal fistula was investigated with various serological techniques. Two aerobic bacterial species, E. dispar and P. mirabilis, and four strict anaerobic bacterial species, B.

fragilis ss. fragilis, F. **varium** and two different strains of C. perfringens, were isolated from fistula secretion of the patient. These strains were used as antigens for tube agglutination, passive hemagglutination, indirect immunofluorescence and immune hemolysis assays with serum specimens obtained before and after operation of the patient. Immune responses were demonstrated to the aerobic as well as to the anaerobic bacterial strains isolated from the patient's fistula. In connection with the operation an active immune response was demonstrated to the aerobic and anaerobic bacterial isolates. Antibodies belonging to IgG and IgA took part in the active immune response while IgM was very little involved. Antibodies responsible for passive hemagglutination reactions were resistant to treatment with beta-mercaptoethanol. Antibodies to aerobic and anaerobic Gram-negative rods were shown to have complement fixing activity. The importance of the demonstrated antibodies for the host's defence against normal intestinal microorganisms and the inflammatory reaction as a consequence of chronic antigenic stimulation in the diseased part of the intestine in patients with Crohn's disease is discussed.

L246 ANSWER 110 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 79197576 EMBASE
DOCUMENT NUMBER: 1979197576
TITLE: An animal model of intra-abdominal sepsis.
AUTHOR: Bartlett J.G.; Gorbach S.L.
CORPORATE SOURCE: Infect. Dis. Serv., Boston VA Hosp., Boston, Mass. 02111, United States
SOURCE: Scandinavian Journal of Infectious Diseases, (1979) 11/SUPPL.19 (26-29).
CODEN: SJIDB7
COUNTRY: Sweden
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
004 Microbiology
048 Gastroenterology
049 Forensic Science Abstracts
LANGUAGE: English

L246 ANSWER 111 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 78391260 EMBASE
DOCUMENT NUMBER: 1978391260
TITLE: Lessons from an animal model of intra-abdominal sepsis.
AUTHOR: Bartlett J.G.; Onderdonk A.B.; Louie T.; et al.
CORPORATE SOURCE: Infect. Dis. Res. Lab., VA Hosp., Boston, Mass., United States
SOURCE: Archives of Surgery, (1978) 113/7 (853-857).
CODEN: ARSUAX
COUNTRY: United States
DOCUMENT TYPE: Journal
FILE SEGMENT: 009 Surgery
004 Microbiology
LANGUAGE: English
AB Intra-abdominal sepsis that involves multiple aerobic and anaerobic bacteria derived from the colonic flora was studied in Wistar rats to determine the relative roles of various microbial species. The rats challenged with pooled colonic contents showed a biphasic disease. Initially, there was acute peritonitis, Escherichia coli bacteremia, and high mortality. In rats that survived this acute peritonitis stage, intra-abdominal abscesses developed, and anaerobic bacteria were the preponderant organisms. Subsequent experiments showed that antibiotics directed against coliforms prevented mortality, whereas agents active

against anaerobes reduced the incidence of abscesses. Challenges with Escherichia coli alone produced bacteremia and death, whereas pure cultures of Bacteroides fragilis caused intra-abdominal abscesses. These observations suggest that both coliforms and anaerobes are important pathogens in intraabdominal sepsis, although the different types of microbes appear to play distinctive roles in the sequence of pathological events.

L246 ANSWER 112 OF 120 CABA COPYRIGHT 2002 CABI
ACCESSION NUMBER: 79:104431 CABA
DOCUMENT NUMBER: 782219157
TITLE: Studies on **Fusobacterium** species in the rumen of cattle. I. Isolation of genus **Fusobacterium** from rumen juice of cattle
AUTHOR: Wada, E.
CORPORATE SOURCE: Dep. Bact., Gifu Univ. Sch. Med., Tsukasa-machi, Gifu-shi, Gifu 500, Japan.
SOURCE: Japanese Journal of Veterinary Science, (1978) Vol. 40, No. 4, pp. 435-439. 13 ref.
DOCUMENT TYPE: Journal
LANGUAGE: Japanese
SUMMARY LANGUAGE: English
AB The nine Fusobacterium species (54 strains) isolated in a modified FM medium from the rumen fluid of 46 cattle were identified as **F. varium** (the predominant species), F. naviforme, F. russii, F. symbiosum, F. necrophorum, F. planti, F. aquatile, F. mortiferum and F. necrogenes Greater numbers of **F. varium** were found in beef cattle on a high carbohydrate diet than from dairy cattle.

L246 ANSWER 113 OF 120 MEDLINE
ACCESSION NUMBER: 78122121 MEDLINE
DOCUMENT NUMBER: 78122121 PubMed ID: 629432
TITLE: Immunization of mice against **Fusobacterium** necrophorum infection by perenteral or oral administration of vaccine.
AUTHOR: Abe P M; Holland J W; Stauffer L R
SOURCE: AMERICAN JOURNAL OF VETERINARY RESEARCH, (1978 Jan) 39 (1) 115-8.
Journal code: 40C; 0375011. ISSN: 0002-9645.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197804
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19900314
Entered Medline: 19780426
AB Immunization of mice against Fusobacterium necrophorum infection was attempted by using 3 vaccination procedures: (1) intraperitoneal (IP) injection of F necrophorum cells in saline solution, (2) IP injection of cells with added aluminum hydroxide adjuvant, and (3) feeding of a powdered mouse diet containing lyophilized cells. One or 2 weekly IP injections of the bacteria cells (in saline solution) for 3, 6, or 12 weeks resulted in protection of 48.7% to 64.5% of the mice against challenge exposure. Of the 2 control groups (given saline solution only), 100% and 97.4% became infected. Weekly IP injections of bacterial cells in an aluminum hydroxide adjuvant for 3, 6, or 12 weeks resulted in protectivity of 54.1% to 77.5%. Of the control mice (given adjuvant only), 97.5% became infected. Bacterial cells fed to mice at a dose level of 1.5 mg (dry weight)/g of powdered diet for 30 days (4 or 5g of diet each day)

resulted in only a delay in the mean time of death as compared with the rapid death of the control mice. The feeding dose of 0.15 mg of cells/g of diet did not delay the mean time of death.

L246 ANSWER 114 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 78271159 EMBASE
DOCUMENT NUMBER: 1978271159
TITLE: Biologic activity of chloramphenicol in experimental intraabdominal sepsis.
AUTHOR: Louie T.J.; Onderdonk A.B.; Bartlett J.G.
CORPORATE SOURCE: VA Hosp., Boston, Mass., United States
SOURCE: Annals of the Royal College of Physicians and Surgeons of Canada, (1978) 11/1 (57).
CODEN: RYPAAO
COUNTRY: Canada
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
LANGUAGE: English

L246 ANSWER 115 OF 120 MEDLINE
ACCESSION NUMBER: 78177868 MEDLINE
DOCUMENT NUMBER: 78177868 PubMed ID: 349444
TITLE: Failure to induce in rabbits effective immunity to a mixed infection of **Fusobacterium** necrophorum and Corynebacterium pyogenes with a combined bacterin.
AUTHOR: Cameron C M; Fuls W J
SOURCE: ONDERSTEPOORT JOURNAL OF VETERINARY RESEARCH, (1977 Dec) 44 (4) 253-5.
Journal code: OI6; 0401107. ISSN: 0030-2465.
PUB. COUNTRY: South Africa
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 197807
ENTRY DATE: Entered STN: 19900314
Last Updated on STN: 19970203
Entered Medline: 19780724
AB Failure to induce in rabbits effective immunity to a mixed infection of Fusobacterium necrophorum and Corynebacterium pyogenes with a combined bacterin. Onderstepoort Journal of Veterinary Research, 44 (4), 253--2;6 (1977). Rabbits were immunized with alum-precipitated, oil adjuvant and an untreated bacterin composed of F. necrophorum and C. pyogenes. Immunized rabbits were challenged intradermally with a mixture of F. necrophorum and C. pyrogenes. Immunized rabbits were challenged intradermally with a mixture of F. necorphorum and C. pyrogenes. Initially a low level of initial transient resistance could be demonstrated but a solid immunity could not be established.

L246 ANSWER 116 OF 120 MEDLINE DUPLICATE 7
ACCESSION NUMBER: 77071334 MEDLINE
DOCUMENT NUMBER: 77071334 PubMed ID: 830651
TITLE: Chemical structure of the lipid A component of lipopolysaccharides from Fusobacterium nucleatum.
AUTHOR: Hase S; Hofstad T; Rietschel E T
SOURCE: JOURNAL OF BACTERIOLOGY, (1977 Jan) 129 (1) 9-14.
Journal code: HH3; 2985120R. ISSN: 0021-9193.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals

ENTRY MONTH: 197702
ENTRY DATE: Entered STN: 19900313
Last Updated on STN: 19900313
Entered Medline: 19770226

AB The lipid A component of lipopolysaccharides from Fusobacterium nucleatum Fev 1 consists of beta-1',6-linked D-glucosamine disaccharides, which carry two phosphate groups: one in glycosidic and one in ester linkage. The amino groups of the glucosamine disaccharides are substituted by D-3-hydroxyhexadecanoic acid. The hydroxyl groups of the disaccharide backbone are acylated by tetradecanoic, hexadecanoic, and D-3-hydroxytetradecanoic acids. Part of the ester-bound D-3-hydroxytetradecanoic acid is 3-O-substituted by tetradecanoic acid. Whereas a similar pattern of fatty acids was detected in lipopolysaccharides from two other F. nucleatum strains, the amide-bound fatty acid in F. **varium** and F. mortiferum was D-3-hydroxytetradecanoic acid. The chemical relationships of lipid A from Fusobacteria and other gram-negative bacteria are discussed.

L246 ANSWER 117 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 77007010 EMBASE
DOCUMENT NUMBER: 1977007010
TITLE: Microbial synergy in experimental intra abdominal abscess.
AUTHOR: Onderdonk A.B.; Bartlett J.G.; Louie T.; et al.
CORPORATE SOURCE: VA Hosp., Boston, Mass. 02130, United States
SOURCE: Infection and Immunity, (1976) 13/1 (22-26).
CODEN: INFIBR
DOCUMENT TYPE: Journal
FILE SEGMENT: 048 Gastroenterology
004 Microbiology
009 Surgery
LANGUAGE: English

AB Intra abdominal sepsis was studied in Wistar rats by using four microbial species: Escherichia coli, enterococci, Bacteroides fragilis, and Fusobacterium varium. These organisms were implanted into the peritoneal cavity singly and in all possible dual combinations. Results were evaluated by mortality rates and the incidence of intra abdominal abscesses on autopsy following sacrifice after 7 days. Mortality was restricted to recipients of E. coli, thus implicating coliforms in the acute lethality associated with this experimental model. Intra abdominal abscesses were produced in 61 of 65 (94%) animals that received the combination of an anaerobe and a facultative organism. Abscesses failed to form with any single strain or with E. coli plus enterococci, and they were detected in one 1 of 19 animals receiving B. fragilis plus F. varium. These results suggest that intra abdominal abscess formation is related to synergy between anaerobes and facultative bacteria.

L246 ANSWER 118 OF 120 EMBASE COPYRIGHT 2002 ELSEVIER SCI. B.V.
ACCESSION NUMBER: 75116764 EMBASE
DOCUMENT NUMBER: 1975116764
TITLE: Activity of lincomycin and clindamycin against anaerobic bacteria (Japanese).
AUTHOR: Mochizuki I.; Shimizu Y.; Isogai K.; et al.
CORPORATE SOURCE: Dept. Urol., Gifu Univ. Sch. Med., Gifu, Japan
SOURCE: CHEMOTHER., (1974) 22/6 (1052-1057).
CODEN: CMTTAM
DOCUMENT TYPE: Journal
FILE SEGMENT: 037 Drug Literature Index
030 Pharmacology
004 Microbiology
LANGUAGE: Japanese

L246 ANSWER 119 OF 120 MEDLINE
ACCESSION NUMBER: 74075343 MEDLINE
DOCUMENT NUMBER: 74075343 PubMed ID: 4520619
TITLE: In vivo and in vitro studies on a new peroxide-containing toothpaste.
AUTHOR: Rundegren J; Fornell J; Ericson T
SOURCE: SCANDINAVIAN JOURNAL OF DENTAL RESEARCH, (1973) 81 (7) 543-7.
Journal code: UCQ; 0270023. ISSN: 0029-845X.
PUB. COUNTRY: Denmark
(CLINICAL TRIAL)
(CONTROLLED CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Dental Journals; Priority Journals
ENTRY MONTH: 197403
ENTRY DATE: Entered STN: 19900310
Last Updated on STN: 19970203
Entered Medline: 19740307

L246 ANSWER 120 OF 120 BIOSIS COPYRIGHT 2002 BIOSIS
ACCESSION NUMBER: 1973:30703 BIOSIS
DOCUMENT NUMBER: BR09:30703
TITLE: STANDARDIZED ANTI MICROBIAL DISC SUSCEPTIBILITY TESTING OF **FUSOBACTERIUM**.
AUTHOR(S): KWOK Y Y; SUTTER V L; FINEGOLD S M
SOURCE: Abstr. Annu. Meet. Am. Soc. Microbiol., (1972) 72, 95.
CODEN: ASMACK. ISSN: 0094-8519.
DOCUMENT TYPE: Conference
FILE SEGMENT: BR; OLD
LANGUAGE: Unavailable